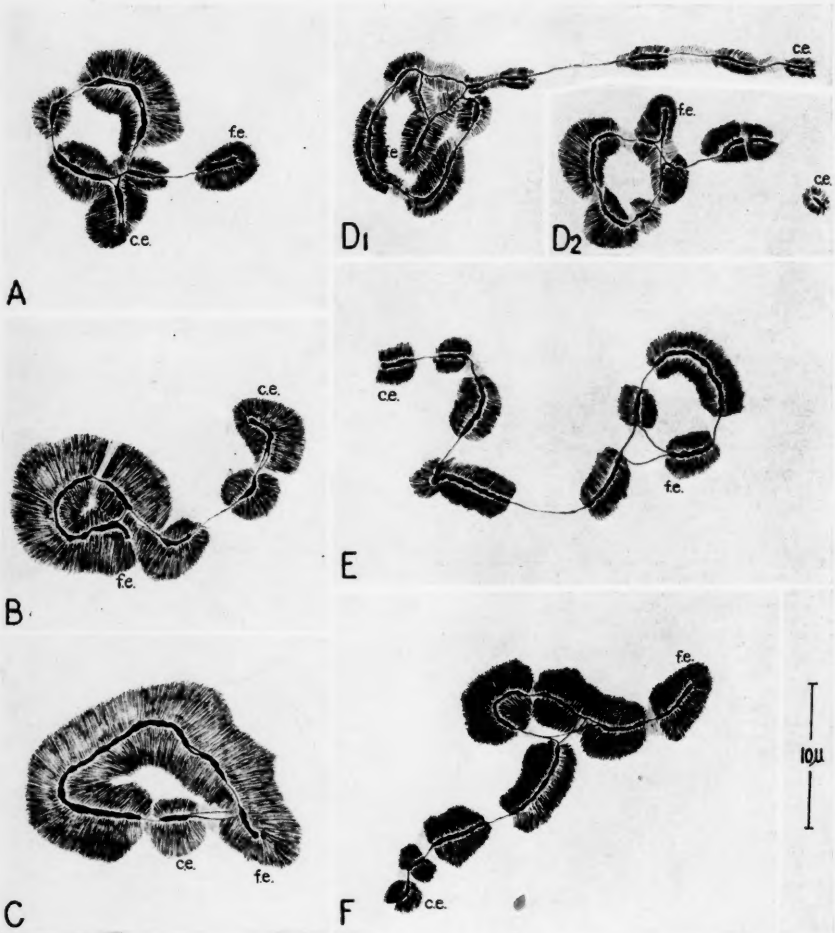
The Journal of HEREDITY



LAMPBRUSH CHROMOSOMES—NEW LEAD TO GENE CHEMISTRY
(SEE PAGE 147)

Polyploidy Induced by Sulphanilamide

A "One-Leaved" Sweet Clover

Mutations in the Mink

VOLUME 32 MAY, 1941 NUMBER 5

The American Genetic Association

An incorporated organization devoted to promoting a knowledge of the laws of heredity and their application to the improvement of plants, animals, and human racial stocks

DAVID FAIRCHILD, President Vice-President, SEWALL WRIGHT Secretary, C. E. LEIGHTY
Treasurer, J. H. KEMPTON
Legal Advisor, STANTON C. PEELLE

EDITORIAL BOARD

Managing Editor, ROBERT C. COOK

M. DEMEREC GEORGE M. DARROW
H. J. MULLER R. R. GRAVES PAUL POPENOE

Members of the Council are ex-officio members of the Executive Board

COUNCIL

W. E. CASTLE, Emeritus Professor of Genetics, Harvard University, and Research Associate of the Carnegie Institution of Washington.
LEON J. COLE, Professor of Genetics, University of Wisconsin.
O. F. COOK, Bionomist in Charge of Breeding Investigations, U. S. Department of Agriculture.
DAVID FAIRCHILD, Agricultural Explorer, U. S. Department of Agriculture.
HARRY V. HARLAN, Senior Agronomist in Charge of Barley Investigation, U. S. Department of Agriculture.
SAMUEL J. HOLMES, Professor of Zoology, University of California, Berkeley, California.
MORLEY A. JULL, Department of Poultry Husbandry, University of Maryland, College Park, Md.
J. H. KEMPTON, Bureau of Plant Industry, U. S. Department of Agriculture.
W. V. LAMBERT, Associate-Director, Purdue University Agricultural Experiment Station, Lafayette, Indiana.
C. E. LEIGHTY, Agronomist in Charge of Dry Land Agriculture, U. S. Department of Agriculture.
MARCUS M. RHOADS, Department of Botany, Columbia University, New York City.
SEWALL WRIGHT, Professor of Zoology, University of Chicago.

ADVISORY COMMITTEE

W. S. ANDERSON, Genetics Department, University of Kentucky, Lexington, Kentucky.
E. B. BABCOCK, Genetics Department, University of California, Berkeley, California.
E. A. BESSEY, Professor of Botany, Michigan State College, Lansing, Michigan.
A. F. BLAKESLEE, Plant Geneticist, Carnegie Institution, Cold Spring Harbor, New York.
W. E. BRYAN, Professor of Plant Breeding, University of Arizona, Tucson, Arizona.
C. B. DAVENPORT, Carnegie Institution of Washington, Cold Spring Harbor, L. I., New York.
RUSSELL W. DUCK, Livestock Editor, *The Rural New Yorker*, Syracuse, N. Y.
R. A. EMERSON, Professor of Plant Breeding, Cornell University, Ithaca, New York.
C. P. GILLETTE, President, The Genetic Foundation, Fort Collins, Colorado.
C. M. GOETHE, Sacramento, California.
HARRISON R. HUNT, Professor of Zoology, Michigan State College, East Lansing.
H. S. JENNINGS, Professor of Zoology, Johns Hopkins University, Baltimore, Maryland.
D. F. JONES, Plant Breeder, Connecticut Agricultural Experiment Station, New Haven, Conn.
M. A. JULL, Senior Poultry Husbandman, U. S. Department of Agriculture, Washington, D. C.
F. B. LINFIELD, Director, Montana Experiment Station, Bozeman, Montana.
C. C. LITTLE, Director, Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine.
JAY L. LUSH, Professor in Animal Husbandry, Iowa State College, Ames, Iowa.
H. H. NEWMAN, Professor of Zoology, University of Chicago, Chicago, Ill.
T. S. PALMER, Expert in Game Conservation, U. S Dept. of Agriculture, Washington, D. C.
JOHN H. PARKER, Professor of Agronomy, Kansas State College, Manhattan, Kansas.
JOHN W. SCOTT, Professor of Zoology, University of Wyoming, Laramie, Wyoming.
A. D. SHAMEL, Physiologist, U. S. Department of Agriculture, Riverside, California.
GEORGE H. SHULL, Professor of Biology, Princeton University, Princeton, New Jersey.
EDMUND W. SINNOTT, Professor of Botany, Barnard College, Columbia University, New York City. (Representative of A. G. A. on Council of Amer. Assoc. for the Advancement of Science.)
W. W. SMITH, Professor of Animal Husbandry, Purdue University, Lafayette, Indiana.
A. B. STOUT, Director of Laboratories, New York Botanical Gardens, New York City.
W. T. SWINGLE, Plant Physiologist in Charge of Breeding Investigations, U. S. Department of Agriculture, Washington, D. C.
W. H. TOMHAVE, American Aberdeen-Angus Breeders' Association, Chicago, Ill.
H. E. WALTER, Professor of Biology, Brown University, Providence, Rhode Island.
H. J. WEBBER, Prof. Subtropical Hort., Citrus Experiment Station, Riverside, California.
E. N. WENTWORTH, Armours' Bureau of Agr'l Research and Economics, Chicago, Illinois.
P. W. WHITING, Zoological Laboratory, University of Pennsylvania, Philadelphia, Pa.

The JOURNAL OF HEREDITY is published monthly by the AMERICAN GENETIC ASSOCIATION at 32nd Street and Elm Avenue, Baltimore, Md., and Victor Building, Washington, D. C. Address all communications regarding editorial matters to Room 308, Victor Building, Washington, D. C. Communications regarding the business management of The JOURNAL OF HEREDITY may be addressed to the publication office, 32nd Street and Elm Avenue, Baltimore, Md., or to the Victor Building, Washington, D. C. Entered as second-class matter February 24, 1923, at the postoffice at Baltimore, Maryland. Acceptance for mailing at the special rate of postage provided for in section 1103, Act of October 3, 1917, authorized December 20, 1918.

Contents copyrighted 1941 by the American Genetic Association. Unauthorized reproduction of material from THE JOURNAL not permitted. The title "Journal of Heredity" is registered as a trade-mark in the U. S. Patent Office.

$3.50 a Year. 35c a Copy. Prices of back numbers on application.

The

Journal of Heredity

Vol. XXXII MAY, 1941 No. 5

CONTENTS

The Structure of the Salivary Gland Chromosomes of Drosophila Melanogaster
By M. Kodani 147

Effect of Sulfanilamide and Other Sulfa Compounds on Nuclear Conditions in Plants
By Hamilton P. Traub 157

A Colchicine Induced Tetraploid Cosmos
By Earl H. Newcomer 161

The Salivary Glands of Prodiamesia Olivacea
By A. M. Melland 165

A Heritable Female-Sterile Plant in Cotton
By G. N. Stroman 168

On the Inheritance of Ear Pit
By E. S. McDonough 169

A New Mutant Leaf Character in Sweet Clover
By E. E. Hartwig 171

The Inheritance of Three Coat Color Mutations in Ranch-Raised Minks
By Sedgwick E. Smith et al 173

NOTES AND REVIEWS

Death Control and Birth Control 156
Rare Forms of Inherited Mental Defect 168
The "Dresden," A Psycho-Lethal 172

STUDENT SUBSCRIPTIONS

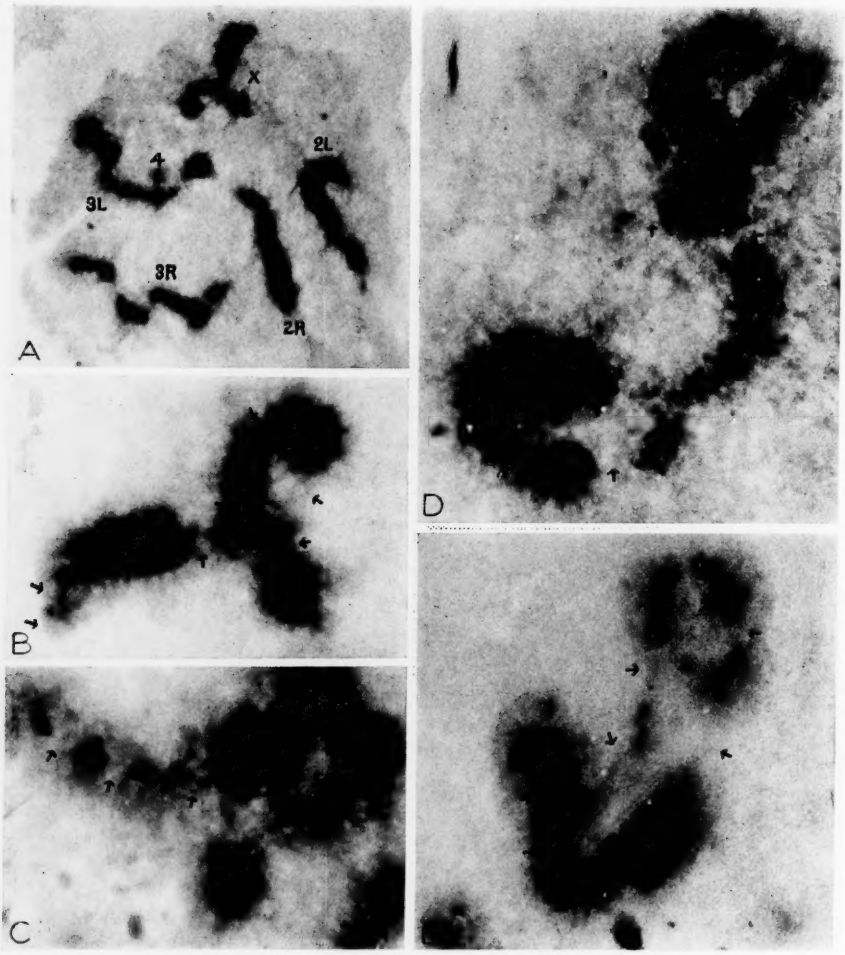
Members engaged in teaching genetics or related subjects are cordially invited to use the JOURNAL as a supplementary text in their classes. The rapid progress of the science, after being so long neglected, is one of the most important and attractive features to place before students. Even the latest textbooks are soon outdated, but they become more interesting as the historical background of the discoveries described in the JOURNAL OF HEREDITY.

Student subscriptions covering the academic year of eight months, in groups of ten or more, may be ordered by members at one dollar ($1.00) each, four months for 50 cents. These special rates to students will be accepted only when applied for by members of the Association, in whose classes the Journals are to be used. The *student subscriptions* will be sent in bulk to the member in whose name they are ordered.

Special Trial Offer to Members of A. G. A.

Members engaged in teaching will be supplied free up to twelve copies of two issues for classroom use. Try the JOURNAL for two months at no expense and under no obligation to continue. Issues for this purpose will be sent in bulk to members applying for them. Send orders to

AMERICAN GENETIC ASSOCIATION
308 VICTOR BUILDING WASHINGTON, D. C.



PHOTOMICROGRAPHS OF THE LAMPBRUSH CHROMOSOMES

Frontispiece

A—A whole female nucleus with all the chromosomes. Note absence of chromocenter. *B*—The *dl-49* inversion. *C*—The *AM* inversion. *D*—The *Bld* translocation with the inversions, *Cl* (above) and *Cy* (below). The translocation is shown by a small dot directing to the right between the inversions. *E*—The rst^3 inversion. The arrows point to the "achromatic" regions. Compare these figures with those of Figure 2. (Same magnification as Figure 2.)

THE STRUCTURE OF THE SALIVARY GLAND CHROMOSOMES

Of *Drosophila Melanogaster*

I—The Eu- and Heterochromatin

M. KODANI

*Department of Zoology, University of California, Berkeley**

IN a recent publication[1] the transformation by chemical treatment of the normal structure of salivary gland chromosomes into that of the lampbrush type was described. It has been shown by further work that the lampbrush chromosome consists of two types of regions which differ in their affinity for chromatin dyes. One of them, which we shall call the "chromatic" region, is very intensely stainable. The other one, called the "achromatic" region, stains only very faintly or sometimes not at all.

These regions have proven to be constant for each chromosome, and they have been closely localized for one of the chromosomes. The purpose of this paper is to report on the method of making these regions visible in lampbrush chromosomes and of localizing them on the salivary map. A discussion of their genetic importance is also included.

Technique

The reagent is prepared by mixing equal amounts of saturated aqueous solutions of urea and 1-N NaOH. The urea solution is made by dissolving 120 grams of urea (c.p.) in 100 cc. of distilled water, which is warmed to 25° C. if the urea crystals are not completely dissolved. The reagents must be used within about an hour after mixing.

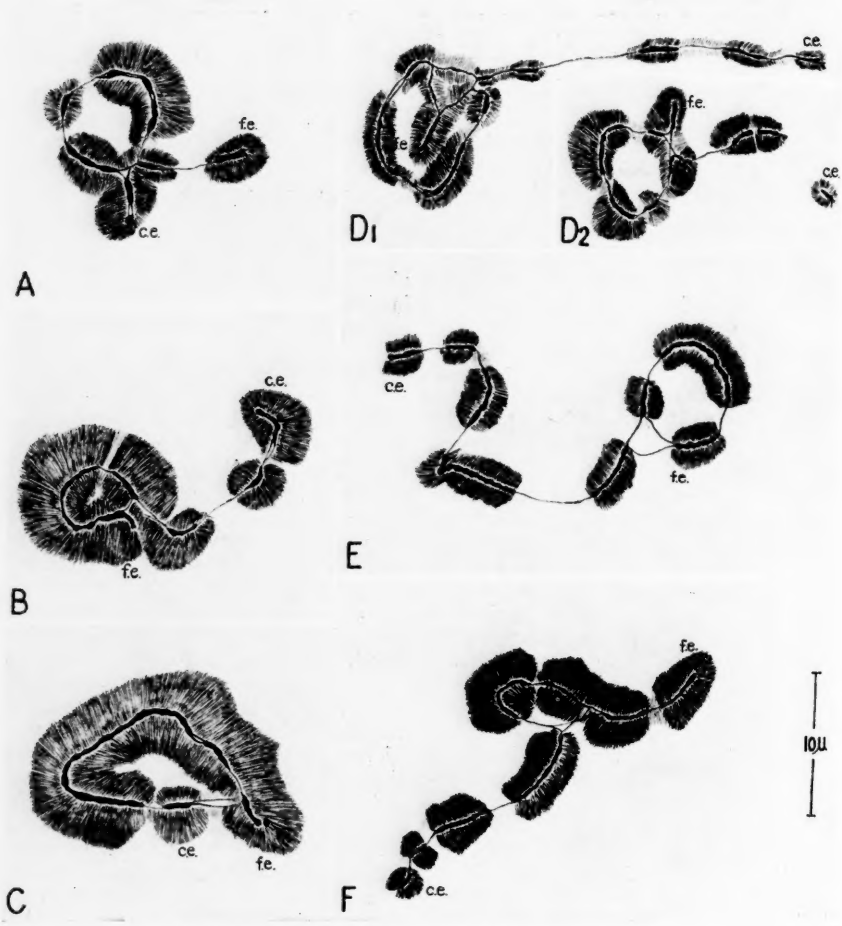
The reagent is applied for thirty minutes directly to glands dissected from mature larvae. The treated glands are stained with acetocarmine for about thirty minutes and then washed thoroughly with 50% aqueous acetic acid before they are smeared. Slides may be made permanent by the vapor-replacement method.[2] The material used in the present study is *Drosophila melanogaster*.

A Brief Description of the Lampbrush Chromosome

A detailed description of the transformation of the normal structure of the salivary gland chromosome to that of the lampbrush type will be given in future publications of this series; here only a few important points will be mentioned so that the material in the following sections can be understood. All the chromosomes treated by the above method are transformed into the lampbrush type. The transformation from the normal structure to the lampbrush type involves a number of transition stages which were described in the previous paper and will be treated in more detail in future publications. However, a brief outline of the process may be given here. The process is essentially a longitudinal contraction of the normal chromosome with a simultaneous condensation of parts of the bands into the central strings, the chromonemata, and of the remaining parts of the bands into the hairs. The transformation of the bands into the chromonemata and hairs is the result of diffusion of chromatin (nucleic acids) from the part of the band from which the hair is formed and incrustation of this diffused chromatin onto the chromonemata which are not visible in the normal chromosome.

The number of visible chromonemata in a completely condensed lampbrush chromosome is one. Occasionally, however, the synapsed chromosomes sepa-

*The author wishes to express his gratitude to Prof. R. Goldschmidt under whose direction this work has been accomplished.



INVERSIONS IN LAMPBRUSH CHROMOSOMES

Figure 1

Camera-lucida drawings of lampbrush chromosomes with inversions. *C.e.* and *f.e.* designate respectively the chromocenter end and the free end of the chromosomes. *A*—The *Cl* inversion of the *X*-chromosome. *B*—The *Cf* inversion of the left arm of chromosome II (2L). *C*—The *Cy* inversion of 2R. D_1 and D_2—The Payne inversion of the 3L chromosome. *E*—The *C* inversion of 3R. *F*—The Payne inversion of 3R.

rate for a short distance showing two chromonemata and furthermore at such places often one or both chromonemata may split again into two, indicating that in each lampbrush chromosome at least four chromonemata are present. Moreover, an *X*-chromosome of a male nucleus shows clearly two chromonemata wherever a split occurs, again indicating beyond doubt that the lampbrush chromosome has a quadripartite structure. This statement should not be taken, however, to imply that the real number of chromonemata is four; there may be more than four although four is the largest number ever observed. The problem of the real number of chromonemata and their structure will be fully discussed in

a publication which is now being prepared.

The lampbrush chromosome shows, further, a number of "achromatic" regions (Frontispiece and Figures 1-2), which consist of chromonemata and hairs like the "chromatic" regions. In these regions the hairs are much finer than in the "chromatic" regions and are only visible in unstretched or only very slightly stretched chromosomes. When the chromosomes are well stretched the hairs in the "achromatic" regions become invisible, presumably because of the wide separation, the hairs being too fine to be visible alone. The Feulgen test indicates that a small amount of nucleic acids is present in the "achromatic" regions. The region of the normal salivary gland chromosome from which the "achromatic" regions of the lampbrush chromosome are derived have been shown by the same test to contain them in larger amounts although not as much as the "chromatic" regions. It is evident from these results that the chromatin (nucleic acids) is removed by our treatment from the "achromatic" regions and incrusted on the "chromatic" regions.

Small chromatic granules which are not as dark as in the "chromatic" regions have been recognized occasionally in the "achromatic" regions. These granules which are presumably the chromomeres retain the nucleic acids while the other parts of the "achromatic" regions lose them almost completely. The behavior of the nucleic acids of various parts of the chromosome toward our reagent is indeed complicated; and it requires further studies for complete physico-chemical explanation.

In the lampbrush chromosomes the chromocenter disappears completely leaving the *X*, *IV*, and the right and left arms of the second and third chromosomes entirely separated from each other (Frontispiece *A*). The *Y*-chromosome does not show up after treatment.

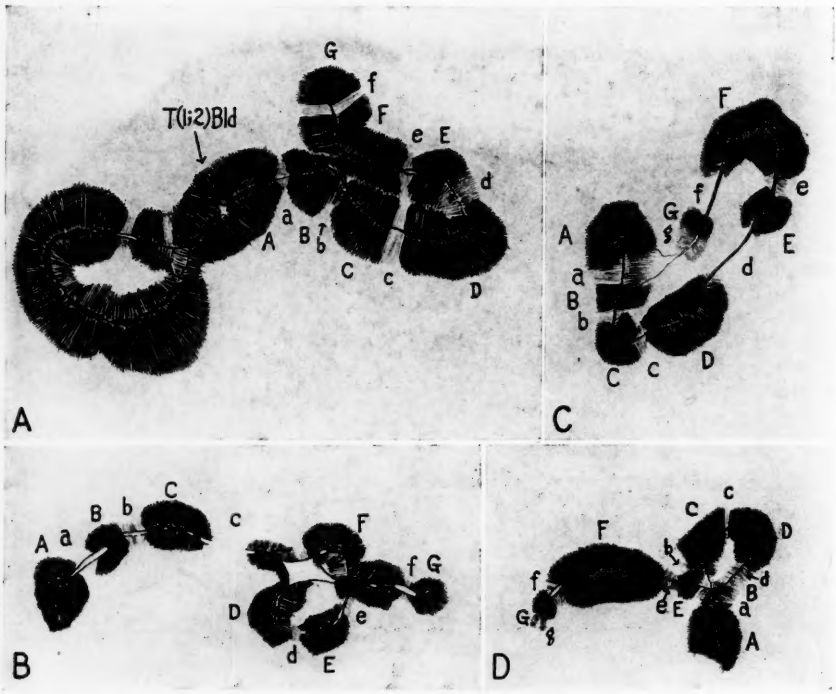
Identification of Lampbrush Chromosomes

The lampbrush chromosomes cannot be identified by their size or shape with the exception of the tiny fourth chromosome, because the separated arms of the second and third chromosomes and the *X*-chromosome are all about equal in length and shape. They can be readily identified, however, by marking them with large inversions or translocations. (Deficiencies and duplications of usual size fail to show up in the lampbrush chromosome and hence cannot be used for marking.) The inversions used for this purpose are (1) *Cl* for the *X*-chromosome, (2) *Cy* (right arm) and *Cy* (left arm) for the right and left arms of the second chromosome, respectively, and (3) Payne (right arm) and Payne (left arm) for the respective arms of the third chromosome. These inversions are shown in Figure 1 as they appear in lampbrush chromosomes. In this figure the characteristic feature of "chromatic" and "achromatic" regions in each arm and chromosome is readily seen, and the constancy of these regions has been proven by comparing many chromosomes.

Both ends of the lampbrush chromosome look alike, because the chromocenter is no longer present, but again they can be distinguished by marking either end of the chromosome by inversions or translocations. The free end of the first chromosome and of the right arm of the second chromosome have been identified by the *Bld*-translocation in which these ends are exchanged (Frontispiece *D*, 1*A*, and 2*A*). The free ends of the remaining arms of the second and third chromosomes have been distinguished by means of the proper subterminal inversions as shown in Figure 1. No description of the autosomes is needed here, for the details will be given in future papers of this series.

Localization of "Chromatic" and "Achromatic" Regions in the X-chromosome

After the chromosomes were identified the "chromatic" and "achromatic" regions of the *X*-chromosome were localized on the salivary map.[3] Two methods, which supplement each other, have been



X-CHROMOSOME INVERSIONS

Figure 2

Camera-lucida drawings of various inversions in the *X*-chromosome (same magnification as Figure 1). Compare these drawings with the corresponding photomicrographs in the Frontispiece. *A*—The *Bld* translocation with inversions *Cl* (right) and *Cy* (left). *B*—The *AM* inversion. *C*—The *rst*[3] inversion. *D*—The *dl-49* inversion. The capital letters indicate the "chromatic" regions and the small letters the "achromatic" regions.

used, since no one method can singly map out all the regions. The first, which will be referred to as the break-point method, is the more accurate of the two; it simply determines the right and left ends of the region according to the break-points of the inversions which are located exactly at those ends. Only a few regions, however, can be localized by this method. Inversions whose points of breakage do not lie exactly at the ends of the regions also help in estimating the positions of the regions in which their break-points are located.

The second method, which will be called the band method, is used to estimate the map length of the region on the basis of its length in the lampbrush chromosome and the rate of its condensation. Every region of the lampbrush chromosome has been found to vary in length presumably due to different amounts of condensation during its formation and also to the varying degrees of stretching which it may undergo at the time of smearing. Therefore, measurements may only be taken from fully condensed and very little stretched chromosomes.

The degree of condensation of the normal chromosome into that of the lampbrush type has been found to be correlated, at least in part, with the number of thick bands contained in the re-

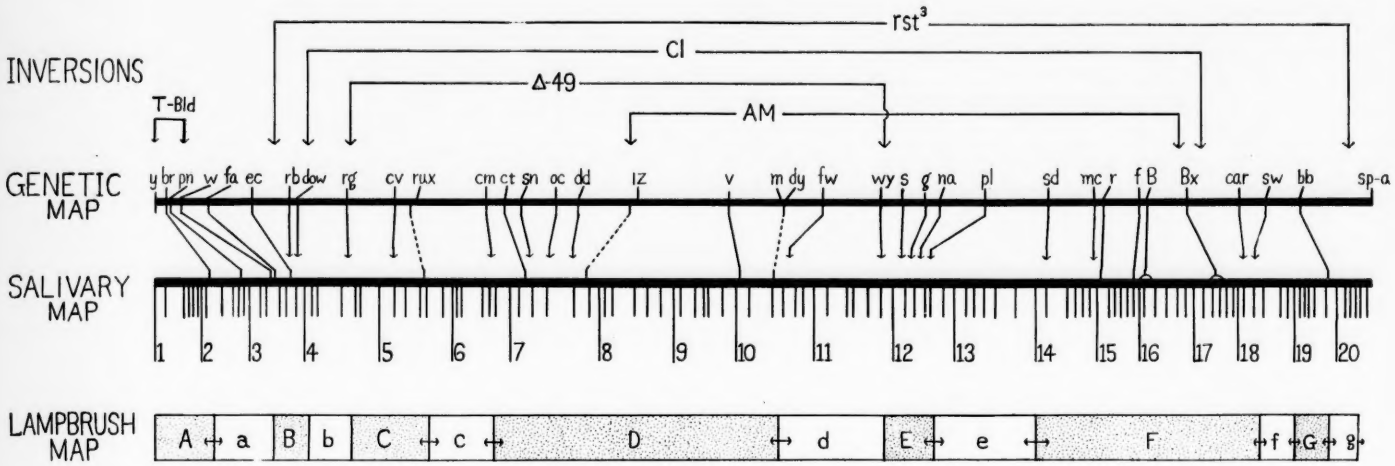
gion. A region of the normal, i.e., untreated, chromosome with a greater number of thick bands shows less condensation than a region with a smaller number of bands of comparable thickness. If the regions were equal in length, then the former would become longer than the latter in the lampbrush chromosome. If, however, their lengths were unequal in the normal chromosome, their relative lengths in the lampbrush chromosome could be estimated on the basis of their lengths and rates of condensation. Similarly, it is possible to estimate their relative lengths in the normal chromosome or in the salivary map by knowing both the lengths in the lampbrush chromosome and the rates of condensation. Once the relative lengths of various regions are determined, their actual extent on the map can be measured from a region whose length on the map is already known by the break-point method. It should be kept in mind that the lengths of the regions determined by this method are only approximations.

Four inversions and one translocation whose points of breakage are properly distributed over the *X*-chromosome have been selected as markers. These are the inversions *Cl, rst,*[3] *dl*-49, and *AM* and the *Bld*-translocation (see Figure 3 for their points of breakage). They are shown in Frontispiece and Figure 2). In Figure 2, the "chromatic" and "achromatic" regions are alphabetically lettered beginning from the free end of the chromosome, the former in capital letters and the latter in small letters.

The regions which can be quite accurately located by the break-point method are the regions "B" and "b." The left limit of "B" is marked by the left point of break of the *rst*[3] inversion (Figure 2*C*) and its right limit approximately by that of the *Cl* inversion (Figure 2*A*). This region, therefore, occupies approximately subdivisions *C, D, E,* and *F* of division 3 and sub-division 4*A*. The region "b" extends from the right end of "B" to the sub-division 4*D* where the left point of break of the *dl-49* inversion is located (Figure 2*D*). Therefore, this region includes approximately the subdivisions *B, C* and a part of *D* of the division 4 (see *Lampbrush map* of Figure 3).

A comparison of these two regions, which are the most accurately localized regions of the chromosome, gives us an idea as to the factors which are involved in the process of condensation. On the salivary map, the region "B" is only slightly longer than the region "b," whereas in the fully condensed lampbrush chromosome such as the one shown in Figure 2*A*, the former is at least twice as long as the latter. In the normal chromosome the region "B" contains approximately twice as many thick bands as the region "b" and since this is the only recognizable difference between these regions, the different degree of condensation is attributed to the difference in the number of the thick bands which are present in them.

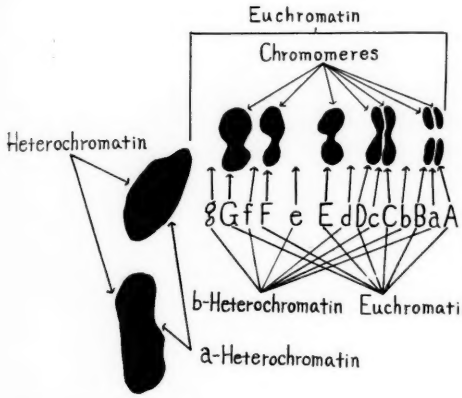
The area between the free end of the chromosome and the left end of the region "B" is occupied by the regions "A" and "a." The boundary line between these regions is estimated by the band method. In a fully condensed lampbrush chromosome such as the one shown in Figure 2*A*, the region "A" is slightly longer than the region "a," but these lengths probably do not represent their true proportion in the normal chromosome, because approximately one-third of the left end of the area is crowded with very thick bands, whereas the remaining two-thirds contain only a few bands of comparable thickness, the majority being extremely thin. The region "a" would be relatively a little longer than it appears in the lampbrush chromosome on account of its higher degree of condensation. Therefore, the boundary line between these regions should be drawn near the left end of division 2. Furthermore, the extent of the region "A" can be estimated by comparing its length in the lampbrush chromosome and the number of thick bands in the normal chromosome to the length of the already accurately determined region "B" and the number of thick bands it includes. The region "A" is approximately one and a half times as long as


INVERSIONS
rst³
Cl
Δ-49
AM
T-Bld
GENETIC MAP
y br pn w fa ec rb dow rg cv rux cm ct sn oc dd lz v m dy fw wy s g na pl sd mc r f B Bx car sw bb sp-a
SALIVARY MAP
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20
LAMPBRUSH MAP
A a B b C c D d E e F f G g

CHROMOSOME MAPS COMPARED

Figure 3

The genetic, salivary, and lampbrush maps of the *X*-chromosome of *Drosophila melanogaster*. The arrows on the lampbrush map indicate regions which have been only approximately related to the salivary map. See text for details.



INTERPRETATIONS COMPARED

Figure 4

The *X*-chromosome of *Drosophila melanogaster* in mitotic prophase, redrawn from Heitz, magnified, and modified slightly (all seven "chromomeres" are shown here whereas in the original figure the first and second "chromomeres" are too close together to be seen as separate "chromomeres" and likewise the third and fourth "chromomeres"). Heitz' interpretation of the parts of the chromosome is given above the chromosome and that of the author below. See text for details.

the region "B" in the lampbrush chromosome, and the number of thick bands (which are about equal in thickness in both regions) of the former is again approximately one and a half times as large as in the latter. Therefore, region "A" is considered to be one and a half times the length of region "B," and consequently the right limit of "A" is once again located at the left end of division 2 (see *lampbrush map* of Figure 3).

The approximate positions of the regions "D," "d," "E," "F," and "g" have been determined according to the points of breakage of various inversions, which are located within or at the margin of those regions. Their map lengths, however, have had to be estimated by the band method. It may be mentioned that the region "g" appears as a short and faintly chromatic tail after the region "G" and is often difficult to recognize because it may be hidden in the hairs of the region "G." The region "e" is automatically determined by locating the adjacent regions just mentioned. The boundary lines of the regions "C" and "c" and of "f" and "G" have been approximately located also by the band method. The procedure used for the localization of all these regions was exactly the same as that which has just been described for the regions "A" and "a." It is not necessary, therefore, to describe it here. The results of the localization are summarized in the *lampbrush map* of Figure 3. The boundary lines whose positions have been approximately located by the band method are so indicated by arrows on either side, and those which have been localized by the break-point method are without arrows, indicating that their positions are accurately fixed.

Discussion

The differentiation of a chromosome into "chromatic" and "achromatic" regions similar to those of the lampbrush chromosome was demonstrated by Heitz[4] in the mitotic prophase of *Drosophila melanogaster*. The *X*-chromosome showed several "chromatic" regions interrupted by "achromatic" parts (Figure 4). Two large "chromatic" regions at the spindle fibre end were called heterochromatic regions and were believed to be genetically inactive. Their existence at this end had previously been pointed out by Muller and Painter.[5] Seven small distal regions (called "chromomeres" by Heitz) were included in the so-called euchromatic region which was believed to contain the active loci.

Heitz also pointed out that each of the seven "chromomeres" is composed of a number of chromomeres, and suggested that each "chromomere" could be separated into smaller chromomeres if the chromosomes were sufficiently stretched as in the salivary gland chromosomes. Our experiments have shown that it is possible to recognize the seven original

"chromomeres" of Heitz after treatment of salivary gland chromosomes with alkali. A close comparison of corresponding "chromomeres" and "chromatic" regions shows, however, that their sizes do not exactly match (see Figure 4 and the *lampbrush map* of Figure 3), but this discrepancy could be attributed to the unequal condensation of the "chromatic" regions after chemical treatment.

The "achromatic" regions of the lampbrush chromosome are undoubtedly different from the "chromatic" regions in regard to the nature of the chromomeres. The difference is considered to be fundamentally genetic; that is, the chromomeres of the "chromatic" regions are considered to be active and capable of mutating whereas those of the "achromatic" regions are not. The terms eu- and heterochromatic may then be used instead of "chromatic" and "achromatic" in order to indicate their genetic distinction. The region which is believed to be totally euchromatic by Heitz would actually consist of eu- and heterochromatic parts (Figure 4). These heterochromatic parts within the euchromatic regions are obviously different not only from the euchromatic parts as regards the nucleic acids but also from the heterochromatic regions at the proximal end of the chromosome which joins the chromocenter and is completely dissolved after treatment. This difference is also shown in the mitotic prophase where the proximal heterochromatic region is deeply stainable whereas the heterochromatic regions of the distal part of the chromosome are not stainable (Figure 4). The proximal and distal heterochromatin are, then, genetically similar, but are cytologically different. In order to indicate the difference and similarity between them, the terms a-heterochromatin and b-heterochromatin will be used respectively for the proximal and distal heterochromatins. The three types of chromatin may be defined as follows: 1. Euchromatin: Genetically active type; present in the thick bands of the normal salivary gland chromosomes and in the regions of the lampbrush chromosomes where the nucleic acids are present in large amounts. 2. a-heterochromatin: Genetically inactive type; exists in the deeply stainable regions of the prophasic chromosomes of mitosis; joined in the chromocenter in the normal salivary gland chromosomes; completely dissolved in the lampbrush chromosomes. 3. b-heterochromatin: Genetically inactive type; exists in the regions of the normal salivary gland chromosomes where a very few or no thick bands are present and in the regions of the lampbrush chromosomes where the nucleic acids are completely or almost completely absent. (See Figure 4.)

The chemical differences of these three types of chromatin are not yet fully understood. The only chemical difference so far known between the eu- and heterochromatin (*a* type) was found by Caspersson.[6] He showed by the ultraviolet absorption method that the euchromatin (including b-heterochromatin) consists of a globulin-like protein whereas the heterochromatin (*a* type) consists of a protein of the histone type. However, the resistance of euchromatin (including b-heterochromatin) to the urea and alkali which constitute our reagent indicates that it does not consist simply of globulin, because globulin is easily dissolved by these chemicals. He reported further that a larger amount of absorption of wave-lengths below 2,600 Å by the euchromatin (including b-heterochromatin) than by the heterochromatin (*a* type) could be most simply explained if free protein substances poor in tyrosine and tryptophane run through the euchromatin but not the heterochromatin. The absence of such substances in the heterochromatin, if such is the case, would account for the peculiar properties of heterochromatin, such as its poor organization in the chromocenter, and its inability to produce localized mutations assuming that localized mutations require such substances.

The interpretation that the "chromatic" regions of the lampbrush chromosomes are euchromatic whereas the "achromatic" parts are heterochromatic is supported by the fact that all but a few known mutant loci fall into the "chromatic" regions (compare the *genetic, salivary* and *lampbrush maps* in

Figure 4). According to this interpretation, then, the mutants whose loci are not yet known on the salivary chromosome could be roughly localized on the salivary map according to the position of the "chromatic" region in which they seem to be included. Their expected approximate loci determined thus are shown in the *genetic map* of Figure 3 by the arrows pointing toward the *salivary map* (the lines connecting the two maps indicate the loci of the genes already localized).

It may be added that on the genetic map there are places which include relatively large genetic units without mutant loci and that most of them are localized in the b-heterochromatic regions of the lampbrush map, indicating that these regions of the chromosomes are not capable of mutating. The *Y*-chromosome and a-heterochromatic regions of the other chromosomes are also known to be incapable of mutating and, as shown above, they are completely dissolved after our treatment leaving no trace of the chromonemata. Whether or not the a-heterochromatin actually lacks chromonemata cannot be decided here. Prokofyeva-Belgovskaya[7] claimed that the chromonemata are present in the *Y*-chromosome and that they are homologous with those of map division 20 of the *X*-chromosome. The most of this part of the *X*-chromosome corresponds to the last *b*-heterochromatic region "g" in the lampbrush map and is not the chromocenter proper. Therefore, if the *Y*-chromosome is actually homologous with this region, it should appear as an "achromatic" lampbrush structure after treatment, but a careful search of *XY*, *XXY*, and *T(y:2)J* has failed to reveal any structure that may be the *Y*-chromosome. The author, therefore, is inclined to doubt the homology of the *Y*-chromosome with division 20 of the *X*-chromosome, but rather regards the former to be completely a-heterochromatic (chromocentral). Now, if the a-heterochromatin is found to lack chromonemata, its behavior toward our reagent would be explained easily and its inability to mutate might be regarded as due to this absence of chromonemata, assuming that the presence of chromonemata is required for localized mutations (the author has in mind Dr. Goldschmidt's theory of chromatin rearrangement).[8]

Furthermore, it should be noted that some heterochromatic regions are not entirely devoid of active loci. One such region is the region "a" which contains the locus of prune (*pn*) and perhaps that of white (*w*). Prune is known to show mottling of the eyes. This phenomenon has been considered as due to the contact of the loci with "inert" substance.[9] The mosaic effect of prune might, therefore, be explained by its location in a heterochromatic region.

Various problems such as the localization of the a- and b-heterochromatin in the autosomes, the structure of the normal salivary gland chromosome and its relation to that of the lampbrush chromosome, and others which were not fully discussed in this paper will be treated in detail in subsequent papers of this series.

Summary

1. A mixture of NaOH and urea was used to produce "chromatic" and "achromatic" regions in the lampbrush chromosomes from the normal chromosomes of salivary glands of *Drosophila melanogaster*.

2. The process of transformation of the normal salivary gland chromosomes to those of the lampbrush type was briefly described. This process involves the longitudinal contraction of the whole chromosome with a simultaneous condensation of parts of the bands into chromonemata and of the remaining parts into hairs. At the same time the lampbrush structure is formed, the nucleic acids diffuse from certain parts of the chromosome forming the "achromatic" regions in the final lampbrush chromosome. These regions were shown by the Feulgen test to contain very little, if any, nucleic acid.

3. Each lampbrush chromosome was identified by marking it with an inversion.

4. The "chromatic" and "achromat-

ic" regions were proven to be constant for each chromosome and they were localized on the *X*-chromosome by special methods. A lampbrush map was then constructed to show the position and extent of all the regions of this chromosome.

5. The "chromatic" regions of the *X*-chromosome were then found to correspond to the "chromomeres" shown by Heitz in the mitotic prophase and were considered to contain the active loci (euchromatic region). The "achromatic" regions were, on the other hand, considered to be heterochromatic. The heterochromatic regions within the euchromatic regions were shown to differ from those at the proximal ends of the chromosomes and in order to indicate their genetic similarity and cytological difference the former were named b-heterochromatin and the latter, a-heterochromatin.

6. On the basis of the interpretation that b-heterochromatin is devoid of active loci, the mutant loci of the *X*-chromosome whose positions are not yet determined on the salivary chromosome were approximately localized on the salivary map.*

AUTHOR'S POSTSCRIPT: After this paper was sent to press a paper by Prokofyeva-Belgovskaya on the inert regions of the *X*-chromosome of *Drosophila melanogaster* was received (*Bulletin De* L'Academie *Des Sciences De L'URSS*, 1939, 363-370). She discovered that the inert regions of the salivary gland chromosomes exhibit a tendency to conjugate with each other as well as with the inert region of the proximal end of the chromosome. She found that there are a number of such regions in the *X*-chromosome and that they are all represented by very faintly stained bands. Prokofyeva-Belgovskaya's inert regions all correspond to those found by the author with the exception of a few extremely short ones. These are probably unrecognizable after our treatment on account of their extreme shortness.

Literature Cited

1. CALVIN, M., M. KODANI, and R. GOLDSCHMIDT. *Proc. Nat. Acad. Sci.* 26:340-349. 1940.
2. BRIDGES, C. B. *D. I. S.* No. 6. 1936.
3. ————. *J. Hered.* 29:11-13. 1938.
4. HEITZ, E. *Zeits. Zellforsch, u. Mik. Anat.* 20:237-287. 1933.
5. MULLER, H. J., and T. S. PAINTER. *Z. I. A. V.* 62:316-365. 1932.
6. CASPERSON, T. *Naturwiss.* 28:514-515. 1940.
7. PROKOFYEVA-BELOVSKAYA, A. A. *Genetics* 22:94-103. 1937.
8. GOLDSCHMIDT, R. *A. A. A. S. Publ.*, 14: 56-66. 1940.
9. SCHULTZ, J. *Proc. Nat. Acad. Sci.* 22: 27-33. 1936.

---

*Services rendered by the Personnel of Works Project Administration Official Project No. 165-1-08-73 are hereby acknowledged.

◆

Death Control and Birth Control

The birth control movement will do well to examine its policies critically. It is unrealistic to suppose that any society will, or should, adopt policies calculated to yield a permanent decline in its numbers. For the present demographic situation, the movement is too single-mindedly occupied with the limitation of fertility. There is need for much more emphasis on the positive aspects of birth control. A program looking to a reduction of the present physical end economic disabilities of parenthood and to the creation of new and wise incentives is a proper concern of the birth control movement. The freedom to be fertile is as important as the freedom to limit fertility in a democratic society that intends to maintain its culture and its stock through a voluntary acceptance of the obligations of parenthood.

Birth control is a major achievement of modern society. Without it the perpetuation of death control and the many other advantages of modern living would have been impossible. But birth control can persist only in a society that places a high value on new life. The United States is in a fortunate position. It has the opportunity to develop such a society, for several decades of growth remain. There is time to observe, to think, and to experiment, but there is no time to be complacent. — STIX and NUTESTEIN — *Controlled Fertility, an evaluation of Clinic Service.* Williams and Wilkins, 1940.

EFFECT OF SULFANILAMIDE AND OTHER SULFA COMPOUNDS

On Nuclear Conditions in Plants

HAMILTON P. TRAUB

U. S. Horticultural Station, Beltsville, Maryland

IN the course of investigations on growth regulating compounds, it was discovered that concentrated solutions of sulfanilamide (P—aminobenzenesulfonylamide) cause polyploidy in plants under the conditions to be described later. Similar activity was observed for other sulfa compounds tested, as sulfa-allantoin, sulfa guanidine, sulfa pyridine (sodium). It was found that the sulfa compounds tested have a growth stimulating effect when used on plants in dilute solution as has been reported for sulfanilamide by Grace.[1]

The effects of concentrated solutions of the sulfa compounds on many plants have been tested in a preliminary way. However, reference is made here only to typical experiments with *Phaseolus vulgaris* and *Allium cepa*.

Typical experiments with the kidney bean, *Phaseolus vulgaris*, on the basis of more than 500 variates, are of interest in this connection. In the bean embryo, in addition to the two single leaves that are already differentiated, the primordium of the first trifoliate leaf is visible. When bean seeds were soaked for six and one-half hours in .5 per cent solution of sulfanilamide and then allowed to remain in a moist condition (solution poured off) over night before planting, germination was somewhat delayed, growth of seedlings was somewhat retarded, and first growth of seedlings was slower as contrasted with controls from seeds soaked in water for the same length of time. Later the plants from treated seeds reached the same height as the controls. These latter had fibrous roots in contrast with the much branched root system of the plants from treated seeds. Such plants from treated seeds when transplanted when fully grown retained their leaves as contrasted with total defoliation for the controls transplanted at the same stage of development.

The above-ground parts of the treated plants showed the characteristic effects of the treatments (Figure 5). In approximately 90 per cent of the plants the leaves were misshapen: on some plants all of the leaflets were fluted and twisted resembling a severe case of virus infection (mosaic). On other plants some of the leaflets and parts of individual leaflets were not fluted or twisted but were of uniform texture and broader as contrasted with the remainder that were characteristically twisted and fluted (Figure 5, *C-D*). In approximately 10 per cent of the plants, the whole portion above the first trifoliate leaf was not fluted and twisted but of uniform texture and on a broader and thicker pattern (Figure 5*B*), and the terminations of of the leaflets were less pointed as contrasted with the acuminate apices in the controls. Fifteen per cent of this latter group of affected plants produced "twinned" fruits (Figure 5*E*). In the 10 per cent of the plants on a broader pattern (Figure 5*B*), the stomata were 30 per cent longer and the pollen grains approximately 10 per cent greater in diameter than in controls (Figure 5*A*); while in the plants with misshapen leaves, the stomata and pollen grains were variable in size, including a large number of more than normal size. Kidney bean cuttings treated at the base for 16 hours with .5 per cent sulfanilamide solution, rooted in sand, and later potted, showed, in most cases, the typical effects described above, in the new tissues formed following treatment (Figure 5*F*, control; and *G*, treated). In the case of the misshapen plants many of the side branches developed normally, but this was not the case for plants with leaves of a broader pattern (Figure 5*B*).

Such responses strongly indicated that

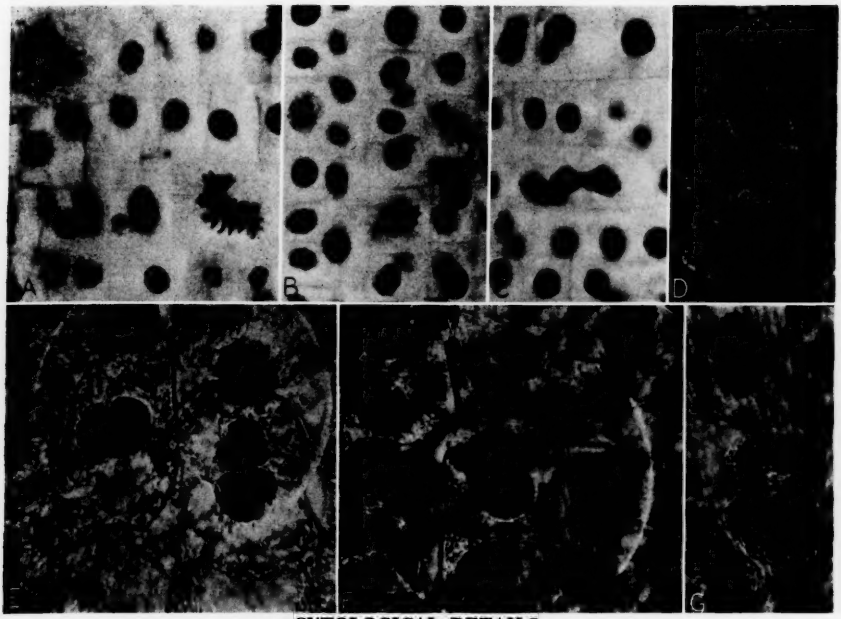
EFFECTS OF TREATING BEANS

Figure 5

Phaseolus vulgaris seed and cutting treatments. *A*—control, plant from seed treated with water. *B* to *E*, incl., plants from seeds treated with .5 per cent sulfanilamide solution; *B*—the entire plant above first trifoliate leaf developed on a broader and thicker pattern; *C*—entire leaflets, and part of some leaflets fluted and twisted; *D*—all leaflets fluted and twisted. *E*—twinned fruits. *F*—control, cutting treated at base with water. *G*—cutting treated at base with .5 per cent sulfanilamide, showing characteristically deformed growth.

polyploidy might be involved in tissues developing after treatment of plants with concentrated solutions of the sulfa compounds. For convenience in cytological observations, *Allium cepa,* the common onion, was used in testing this hypothesis.

Onion bulbs were rooted with bases in water for two days; then lots were transferred to .25 and .5 per cent solutions of sulfanilamide respectively for two days;



CYTOLOGICAL DETAILS

Figure 6

Longitudinal sections of onion (*Allium cepa*) root tip; nuclear conditions in cortex following treatment with .5 per cent sulfanilamide solution. *A*—two tetraploid cells, metaphase, ×600. *B*—two diploid cells, metaphase, ×600. Cells with "dumbell-shaped" nuclei (*C* ×600, *D* ×950). *E*—cells with partially separated nuclei, ×950. *F*—binucleate cells, resting stage, ×950. *G*—binucleate cell, anaphase, ×950.

and then the bases of the bulbs were again placed in water for two days. Within less than 24 hours after the bases of the rooted bulbs were placed in the concentrated solutions, hypertrophy or swelling took place in the region immediately back of the root tip meristem. This swelling is apparently in the region of elongation.

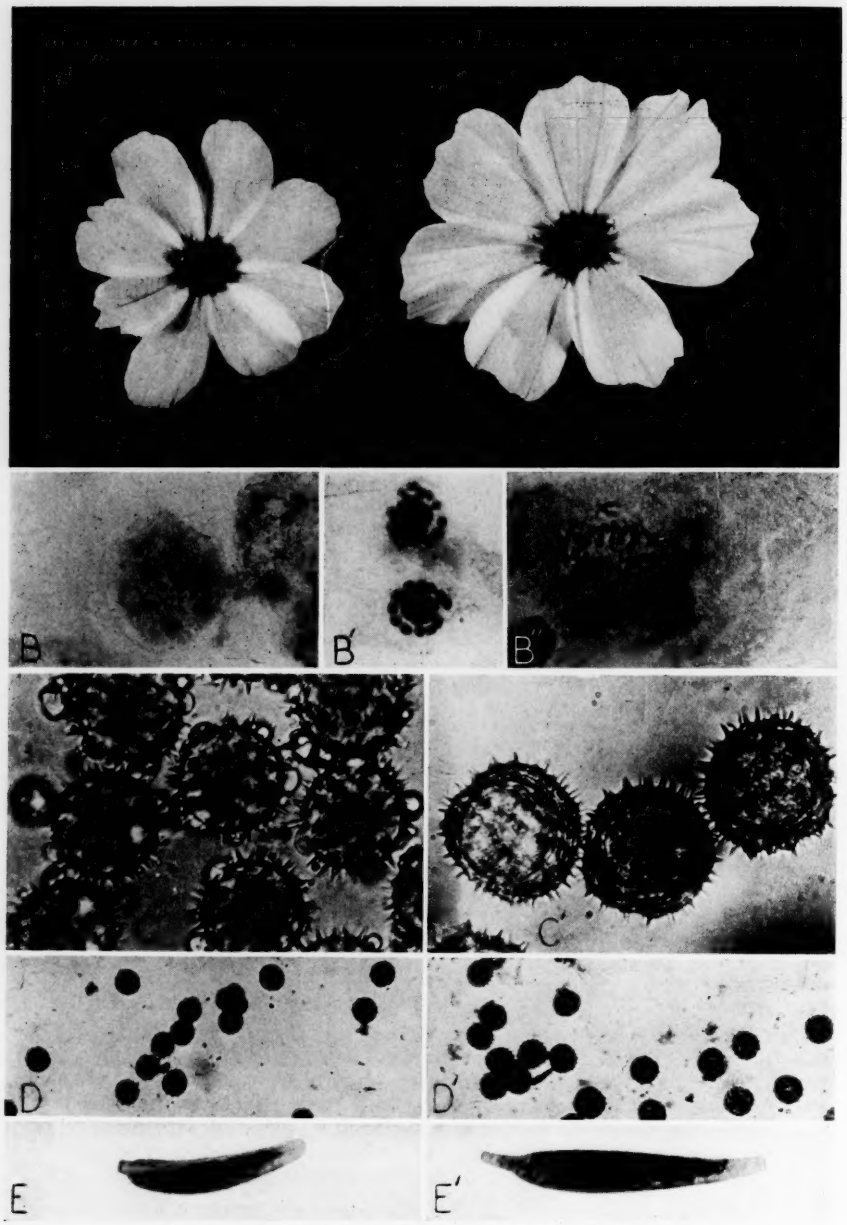
Cytological examination, using the paraffin embedding method and staining with Crystal Violet and iodine, showed that in the new root tissue formed after transferring from water to sulfanilamide solution and then again to water, mitosis, in a part of the cells, was profoundly affected, largely in the cortex. The mitotic abnormalities observed included various nuclear conditions (Figure 6). There were polyploid cells with double (4X) chromosome number (Figure 6 *A*), cells with single large nuclei, cells with "dumb-bell-shaped" nuclei (Figure 6*C-D*), and bi-nucleate cells (Figure 6, *E, F, G*). In the case of material transferred from .25 per cent solution to water, few bi-nucleate cells were found in contrast with numerous cells with two nuclei in roots taken from .5 per cent solution and placed in water. Experiments are under way to determine the mechanism involved in the production of these abnormalities.

It is of interest to note in connection with these results that Eigsti[2] has reported that "mitotic irregularities" were induced in the pollen tube of *Tradescantia occidentalis* with sulfanilamide.

Literature Cited

1. Grace, N. H. Note on sulfanilamide and other chemicals that act as growth promoting substances. *Canad. Jour. Res.* (Sec. C.) 16:142-144. 1938.

2. Eigsti, O. J. A preliminary study of the effects of certain organic substances upon cell division. (Abstract.) *Amer. Jour. Bot.* 27:15s. 1940.



COMPARISONS BETWEEN DIPLOID AND TETRAPLOID

Figure 7

Figures on the left column, *A*, *B*, *C*, *D*, *E*, show the flower, chromosomes, pollen grains, seed, and stamens, respectively, of the normal diploid. Figures *A'*, *B'*, *C'*, *D'*, *E'* show the respective tetraploid counterparts. Magnification: Figs. *A*, *A'* reduced approx. ¼; Figs. *B*, *B'*, *B* , ×750; Figs. *C*, *C'*, ×560; Figs. *D*, *D'*, ×125; Figs. *E*, *E'*, ×5.5.

A COLCHICINE INDUCED TETRAPLOID COSMOS

Some Comparisons with Its Diploid Progenitors*

EARL H. NEWCOMER
University of North Carolina

SINCE the recent introduction of colchicine in this country as a specific technic for the production of polyploids in plants, there have already been numerous reports of successful results from its use, and the specific action of the drug upon the cell economy has been studied by several workers. The literature on the subject has been recently reviewed by Dermen.[4] Emsweller and Brierly[6] cite as the most promising of the colchicine-induced tetraploids produced to date the marigolds and snapdragons of Ruttle and Nebel, to which might be added the lilies reported by Emsweller and Brierly and the tetraploid flax of Györffy.[8] That colchicine does not belong to the category of fugacious fads seems certain, and in addition to its apparent practical importance as an aid to the plant breeder, it may be extremely useful to the further understanding of the significance of autopolyploidy in plants.

The effect of polyploidy on cell size and other characters of plants and the relationship of these change to other biological factors have been discussed in papers on polyploidy by Gerassimoff,[7] Navaschin,[13] Müntzing,[12] Hagerup,[9] Anderson,[1] Wettstein,[20] Sax and Sax,[16] Stebbins,[17] Randolph and Hand,[14] Bowden,[3] and many others. From a consideration of this work and the current reports, it appears that generalizations as to the functional significance of, and the morphological characteristics induced by, allo- or autopolyploidy in plants are unsafe and that there is no single criterion which is adequate for the detection of polyploidy in all cases, save a chromosome count. Exceptions have been reported for every one of the usual morphological criteria such as size and distribution of stomata, pollen size, stature, cell size, etc., and there is evidence to suggest that some of the morphological distinctions between diploids and polyploids may not be permanent, reversions in at least one case having been reported by Wettstein[20] in *Bryum*. Similar regression may or may not occur in the higher plants. As H. J. Sax[15] pointed out, "if it should be established that there is a general tendency for polyploids to regress in cell size to that of diploids, the relative time of origin of a polyploid species might be suggested according to the size of its cells to that of the diploid."

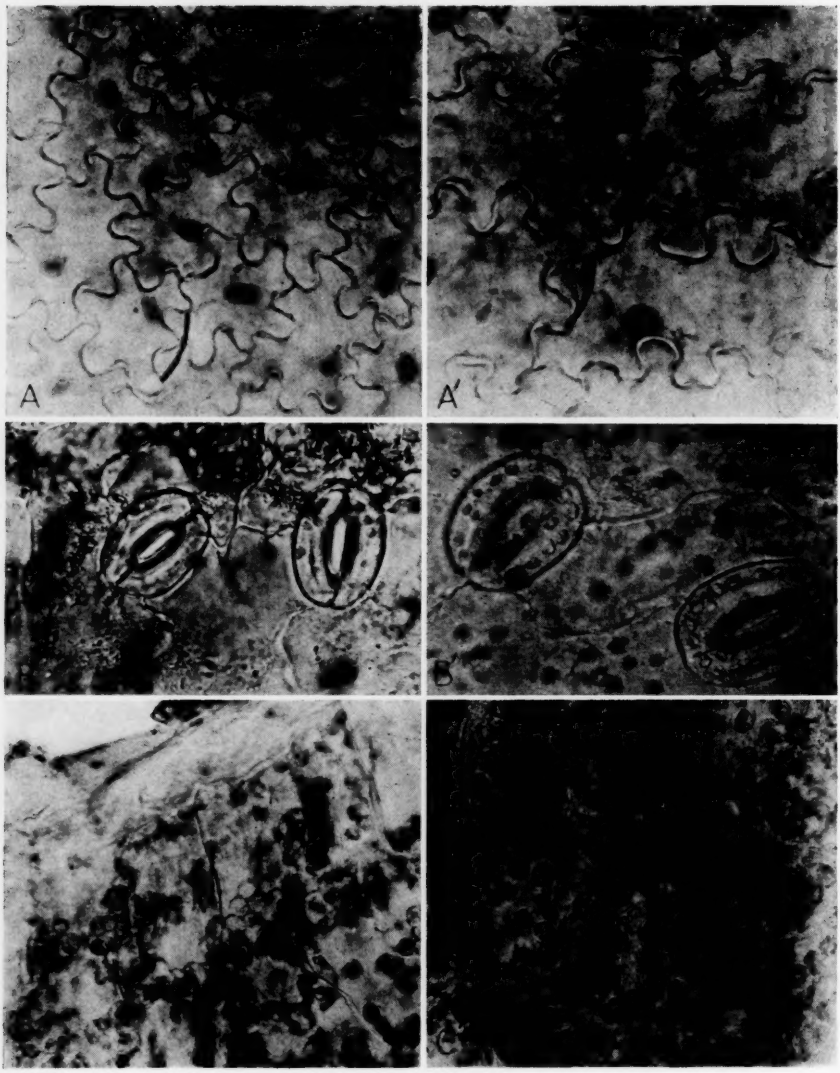
With the appearance of more and new polyploids made possible with the colchicine technic, additional information on this subject should be forthcoming.

Materials and Methods

Seeds of *Cosmos bipinnàtus* Cav., variety Early Sensation, supplied by the Ferry Morse Seed Company, were grown in flats in the greenhouse until the seedlings attained a height of several inches. The apical growing points were treated with various concentrations of colchicine in various solvents and mixtures. The most effective treatment was a .2% solution of colchicine in the emulsion originated by Warmke and Blakeslee.[19] In several instances, two applications of this emulsion with a pipette or atomizer induced polyploidy.

The seedlings which showed hypertrophy, the usual symptom of a polyploid condition, were set out in the field to mature. Seeds of these plants were collected and an F_1 generation grown, from which the materials for the present study were obtained. Chromosome examinations were made on all P_1 plants saved

*Journal Article No. 491, N.S., of the Michigan State College Experiment Station.



TISSUES COMPARED

Figure 8

A, B, C, on the left column show the mature petal epidermal cells, stomata and chloroplasts, respectively, and *A', B', C',* show their respective tetraploid counterparts. Magnification of all figures: ×560.

for seed as a check on preliminary diagnoses. Normal, untreated plants were grown nearby for controls.

The diploid *Cosmos bipinnàtus* has a chromosome complement of twelve pairs, and the autotetraploid twenty-four pairs.

Observations

1. General morphological comparisons:

	Diploid	*Tetraploid*
Flower	62 mm.	81 mm.
Pollen	22 μ	26 μ
Epidermal cells	18 × 30 μ	30 × 62 μ
Stomata	20 × 28 μ	26 × 32 μ
Plastids	4 × 6 μ	5 × 7 μ
Seed	1 × 6 mm.	1.2 × 9 mm.

The F_1 tetraploid Cosmos plants were stockier, slightly bushier, and shorter in stature than their diploid progenitors and controls. Flowers of the tetraploid were larger (Figure 7 *A*, *A'*) and were produced in similar abundance. A comparison of seed germination and growth of the seedlings of the tetraploids with the diploids demonstrated that the seeds and seedlings of the tetraploid in this instance were slower in initial germination and rate of seedling growth in height. This developmental difference is apparently persistent, the tetraploids coming into flower later but persisting longer than the diploids.

Similarly, in all the other morphological comparisons made in this study the tetraploid showed an advantage in size which was not, however, proportional in all cases. For example, the plastids of the tetraploid were only slightly larger (Figure 8 *C'*), whereas the mature petal epidermal cells (8 *A*, *A'*) and stomata (8 *B*, *B'*) were conspicuously greater in size. Differences in size of the pollen grains were difficult to detect or to measure under low power (Figure 7 *D*, *D'*), although the camera seems to show a rather conspicuous difference. It is interesting to observe that the slight increase in size of the tetraploid pollen grains is not due to an increased volume of cell contents but to the increased thickening of the cell walls (Figure 7 *C*, *C'*). The actual cytoplasmic volume of the pollen grains is no greater, and in many instances is actually less in the tetraploid as compared with the diploid. These observations seem to be exceptions to the generally valid rule that increase in nuclear substance stands in causal relation to cell size and consequently to the general amount of protoplasm.[7, 10].

The generalization of Kostoff[11] that the chloroplasts do not increase, but rather decrease in size with euploid increase of chromosome numbers, was not substantiated in this study (Figure 8 *C*, *C'*). Plastids were larger in the tetraploid Cosmos, but their size was not doubled as Sax and Sax[16] reported for tetraploid *Tradescantia*.

Seeds of the autotetraploid Cosmos were macroscopically and conspicuously larger and heavier than those of the diploids (Figure 7 *D*, *D'*).

Observations on the chromosome behavior of the tetraploid during meiosis revealed normal pairing and disjunction, although there was a variable amount of secondary association apparently between the duplicate bivalents. While occasional anaphase lagging of one to several pairs of chromosomes was infrequently observed, the laggards apparently always reached the poles in time to be included in the telophase nuclei.

Mutations and Sterility

An apparent mutant type appeared in one of the treated P_1 plants, in which the ray florets of the inflorescence were tubular and zygomorphic, and the plant, though a prolific bloomer in the field, was completely sterile. Since some variations of flower type occur normally in this variety, although nothing similar appeared in a large population of controls, it cannot be stated with certainty that the mutation was induced by colchicine. A chromosome examination revealed the mutant to be a tetraploid.

Similar mutant types in colchicine-treated progeny were observed by Thompson and Kosar[18] and Emsweller and Brierly,[6] and the latter authors observed that "it would appear that chromosome changes other than simple reduplication must have taken place." Whether these mutants may be due to

chromosome fragmentation as suggested by the work of Eigsti,[5] further investigation may determine. Additional evidence that colchicine may produce chromosome changes other than simple reduplication has just appeared in a paper by Bergner, Avery and Blakeslee.[2]

Probably many desirable and useful seed-propagated autotetraploids have been lost because of the rather high frequency of sterility found to occur. In our own work, a very promising tetraploid Phlox and an Ageratum were lost because of complete sterility. It was at first thought that the tetraploid Cosmos was similarly sterile, as no seed was ever produced in plants grown in the greenhouse, whereas field-grown siblings produced an abundant yield of seed. It is not suggested that greenhouse conditions are solely or even largely responsible for the high degree of sterility found in colchicine-induced autotetraploids, but they may be a contributing factor.

Summary

The colchicine-induced autotetraploid Cosmos of this study is larger in every morphological comparison made, with the exception of height, than its diploid progenitor. The increase in size of the various units compared was not proportional in all instances. In actual cytoplasmic volume of the pollen grains there is little, if any, measurable difference, and only a slight increase in chloroplast size of the tetraploid is discernible. A pronounced increase in thickness of the cell walls accounted for the slight increase in size of the tetraploid pollen grains. The most obvious results of polyploidy in this study were the increase in size of flower, petal epidermal cells, stomata, and seeds.

Field-grown plants were fully fertile, whereas greenhouse-grown siblings were completely sterile.

Literature Cited

1. Anderson, E. Cytology in its relation to taxonomy. *Bot. Rev.* 3:335-350. 1937.

2. Bergner, A. D., Avery, A. G., and Blakeslee, A. F. Chromosome deficiencies in *Datura* induced by colchicine treatment. *Amer. Jour. Bot.* 27:676-683. 1940.

3. Bowden W. M. Diploidy, polyploidy, and winter hardiness relationships in the flowering plants. *Amer. Jour. Bot.* 27:357-371. 1940.

4. Dermen, H. Colchicine polyploidy and technique. *Bot. Rev.* 6:599-635. 1940.

5. Eigsti, O. J. The effects of colchicine upon the division of the generative cell in *Polygonatum, Tradescantia* and *Lilium*. *Amer. Jour. Bot.* 27:512-524. 1940.

6. Emsweller, S. L. and Brierly, P. Colchicine-induced tetraploidy in *Lilium*. *Jour. Hered.* 31:223-230. 1940.

7. Gerassimoff, J. J. Die Abhängigkeit der Grösse der Zelle von der Menge ihrer Kernmasse. *Zeitschr. Allgem. Physiol.* 1:220-258. 1902.

8. Györffy, B. Durch Kolchizinbehandlung erzeugte polyploide Pflanzen. *Naturwiss.* 26:547. 1938.

9. Hagerup, O. Ueber Polyploidie in Beziehung zu Klima, Oekologie, und Phylogenie; Chromosomenzahlen aus Timbuktu. *Hereditas* 16:19-40. 1932.

10. Kostoff, D. Directed heritable variations conditioned by euploid chromosome alteration. *Jour. Genet.* 36:447-468. 1938.

11. Kostoff, D. and Orlov, A. The size of chloroplasts in eupolyploid forms of *Nicotiana* and *Solanum*. *Ann. Bot. N. S.* 2:883-886. 1938.

12. Müntzing, A. The evolutionary significance of autopolyploidy. *Hereditas* 21:263-378. 1936.

13. Navashin, M. Chromatin mass and cell volume in related species. *Univ. Calif. Publ. Agr. Sci.* 6:207-230. 1931.

14. Randolph, L. F. and Hand, D. B. Relation between carotenoid content and number of genes per cell in diploid and tetraploid corn. *Jour. Agr. Res.* 60:51-64. 1940.

15. Sax, H. J. The relation between stomatal counts and chromosome numbers. *Jour. Arnold Arb.* 19:437-441. 1938.

16. Sax, K. and Sax, H. J. Stomata size and distribution in diploid plants. *Jour. Arnold Arb.* 18:164-172. 1937.

17. Stebbins, G. L. The significance of polyploidy in plant evolution. *Amer. Nat.* 74:54-66. 1940.

18. Thompson, R. C. and Kosar, W. F. Polyploidy in lettuce induced by colchicine. *Proc. Amer. Soc. Hort. Sci.* 36:641-644. 1937.

19. Warmke, H. E. and Blakeslee, A. F. Induction of simple and multiple polyploidy in *Nicotiana* by colchicine treatment. *Jour. Hered.* 30:419-432. 1939.

20. Wettstein, F. von. Experimentelle Untersuchungen zum Artbildungs-problem. 1. Zellgrössenregulation und Fertilwerden einer polyploiden Bryum-Sippe. *Zeitschr. Indukt. Abstamm.-Vererbungsl.* 14:34-53. 1937.

THE SALIVARY GLANDS OF PRODIAMESIA OLIVACEA

A case of asymmetry in the Diptera

A. M. MELLAND

Zoology Department, Cambridge, England

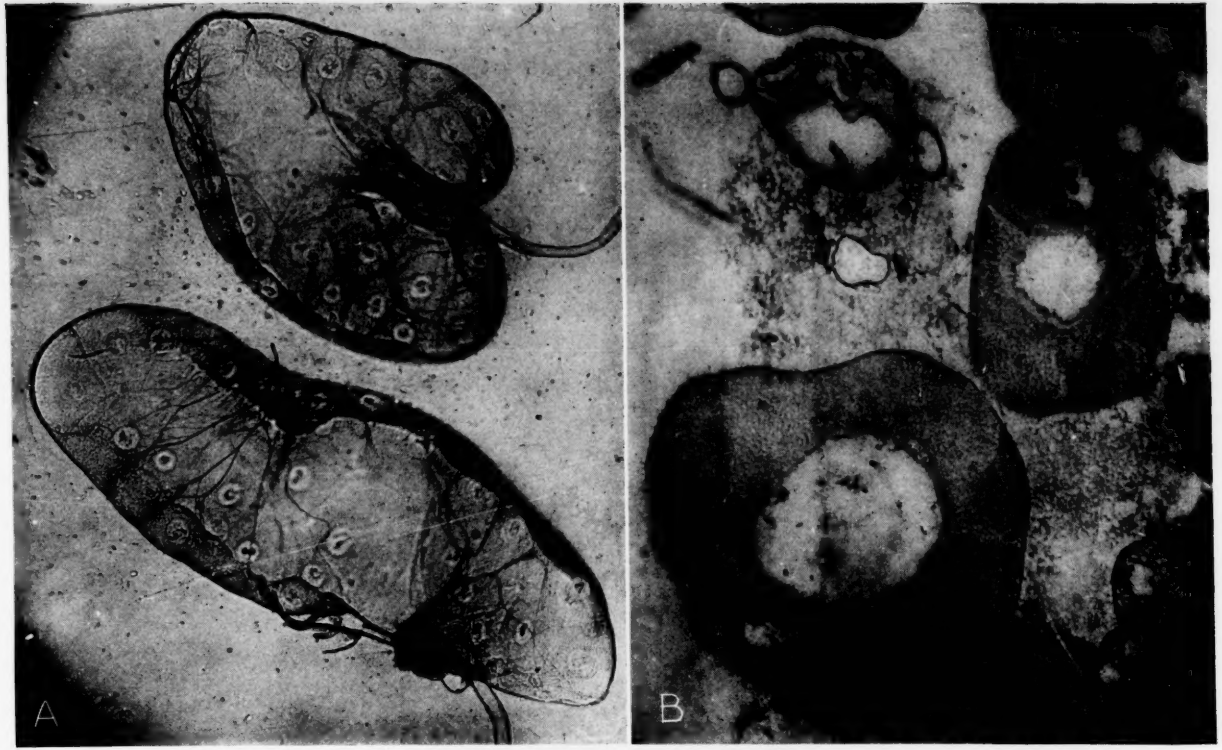
CHROMOSOMES OF PRODAMESIA

Figure 9

Acetocarmine smears of salivary gland chromosomes of *Prodiamesia olivacea*. This species has one long salivary gland chromosome, two of medium length, a fourth short chromosome and a fragment.

THE salivary glands of the larva of *Prodiamesia olivacea* Mg., show a morphological and specific asymmetry. This was confirmed after examination of many specimens from several localities. As there are no other species of *Prodiamesia* in Great Britain it is not known how far this description holds for other species.

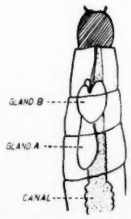
Gland *A* (Figure 11) is long and blimp-shaped while gland *B* is lobed like that of a *Chironomus* larva. There is no obvious difference between the nuclei of the glands, each gland having about forty nuclei. In neither case are the nuclei restricted to one plane as in *Chironomus*. F. W. Edwards,[1] classifying adults, and O. A. Johannsen,[2] in a key of the immature stages, both describe the Diamesinae, of which *Prodiamesia* is a member, as an intermediate group and the glands seem to bear witness to this judgment, for they are intermediate between the football gland of a Tanypod larva and the flat gland of a *Chironomus*. In size they are similar to *Chironomus dorsalis*



GLANDS ENTIRE AND IN SECTION

Figure 10

A—The entire salivary glands of *P. olivacea* photographed in the body fluid. Each of them contains about forty cells, with large nuclei containing the giant chromosomes. Several of the nuclei are clearly visible. The shape and relative size of the glands is a constant characteristic of this species. *B*—Section through the two glands in their normal position, the long gland which projects about 120μ beyond the other being at the bottom; the short lobed gland above it to the right. Three nuclei are visible in this section.



POSITION OF THE GLANDS
Figure 11

The location of the glands in the forepart of the larva of *P. olivacea* is here diagrammed. In most related forms the glands are of the same size and shape.

and the chromosomes are as contracted and separate as easily when smeared: *Tanypus* chromosomes are more like those of *Drosophila*. But, while the Chironominae have one long chromosome and two of medium length, with or without nucleoli, and a short fourth chromosome which invariably has a large nucleolus but varies as to length and degree of dispersion, *Prodiamesia olivacea* resembles the Tanypodinae in having a chromocenter and a fifth fragment which is usually so dispersed as to resemble a star more than a normal salivary gland chromosome.[3]

The tracheae attached to the glands are found also in *Tanypus* but not in *Chironomus*.

Literature Cited

1. Edwards, F. W. British Non-biting Midges. *Trans. Ent. Soc.* 77:279-430. 1929.
2. Johannsen, O. A. Aquatic Diptera. Part 3, Memoir 205. Ithaca, N. Y. 1937.
3. Bauer, H. Beitrage zur vergleichende Morphologie der Speicheldrüsenchromosomen. *Zool. Jahrb. Abt. allg. Zool. Phys.* 56:239-276. 1937.

◆

A HERITABLE FEMALE-STERILE TYPE IN COTTON

G. N. Stroman
New Mexico Experiment Station

A COMMON form of "rogue" plant found in fields of Acala No. 8 cotton is a type that is devoid of bolls. It is easily detected late in the season because it is very tall. Although a large number of flowers are produced, the bolls are all shed when they are about the size of pecans. The taller growth of the plants is probably a direct result of the sterility. Harland[1] states that the variant cotton plants called by the natives of Trinidad "man cotton" are haploids. He showed that the plants do not have viable pollen but are fertile as female parents. Hutchinson and Gadkan[2] report a sterile form in "million dollar" cotton caused by one gene.

In the regular cotton-breeding progeny test at the New Mexico Experiment Station in 1936, there appeared in one progeny from an individual plant selection from Acala 8, sixteen sterile plants in a total of 42. Reciprocal crosses were made between these sterile plants in row 1840 and other plants of other rows adjacent to it. Where the sterile plant was used as the female parent, no seed was set but where it was used as the male parent, a good set of seed was produced. The pollen of these plants was microscopically examined and while there were a few shrunken pollen grains, most of the grains appeared to be normal.

The crosses made were planted the following year, but coupled with an apparent mixup in planting and poor germination they were not carried further. At the time the writer was more interested in how to eliminate this type from the breeding plots than in its physiology.

The normal plants of the number 1840 row were planted and some were followed through to 1939. A record* of

*Tables 1-3 have been issued by us through Auxiliary Publication, and may be obtained from the nonprofit Bibliofilm Service, care U. S. Department of Agriculture Library, Washington, D. C., by ordering Document 1509, remitting 25 cents for copy in microfilm, readably enlarged full-size on reading machine or hand viewer, or 50 cents for copy in the form of paper photoprints readable without mechanical aid.

these is found in Tables I, II and III.

In Table I is shown the 1937 counts of segregation of 24 normal plants of 1936 as to normal and sterile plants and as to the mean heights of normals and steriles. The totals of the segregating progenies gave 771 normals to 210 steriles, which approximates a 3:1 relationship. The reason these progenies were continued in 1938 and 1939 was to test the possibility that more than one factor pair was involved. The 1939 planting consisted of one family which appeared to have a tendency to a 9:7 ratio, one family which approached a 13:3 ratio, and one with a ratio of 3:1. However, the 1939 counts show that while in two of the families the total of normals vs. steriles do not fit very closely, the number of heterozygous progenies to homozygous ones do tend to confirm the hypothesis that one factor only is operating with respect to this sterile character.

The counts in 1938 are shown in Table II and those in 1939 in Table III, and all confirm the existence of one basic factor. In 1937, there were eight non-segregating progenies and 17 segregating progenies; in 1938, five and nine of the 2592 family, and four and ten of the 2594 family; and in 1939 there were six non-segregating and 12 segregating progenies of the 2960 family, six to 19 in the 2977 family, and 10 to 19 in the 2778 family. The total counts of segregating families shown at the bottom of each table approximate a 3:1 ratio. Since all progenies came from open-pollinated seed, it is not surprising that better ratios were not obtained.

In the case of practical plant breeding, pedigree planting will very promptly eliminate this type of sterile rogue. Since the sterile plants produce viable pollen in profusion, there would be some crossing where seed was used that was not self-pollinated. It is difficult to identify the steriles until midseason, when they can be pulled out, but in the roguing of registered and certified fields the harm of crossing has usually occurred before they can be located and removed.

Literature Cited

1. Harland, S. C. Haploids in polyembryonic seeds of Sea Island Cotton. *Jour. Hered.* 27:229-231. 1936.

2. Hutchinson, J. B. and Gadkan, P. D. A note on the inheritance of sterility in cotton. *Indian Jour. Agr. Sci.* 5(5):619-623. 1935.

◆

Rare Forms of Inherited Mental Defect

Another very interesting recessive disease, which causes gross mental retardation, but which is not progressive, was identified in Norway a few years ago by a biochemist, Fölling. He found that a substance, phenylpyruvic acid, was always present in the urines of certain imbeciles; in the normal person this substance is not excreted at all, possibly because it is utilized in the functioning of the brain. The disease seems to be more widely spread in Norway than in some other countries, though cases have been described in England, Scotland and France. In the United States many cases have been found, but only a small number of them have Norwegian ancestry. Other important recessively determined types of mental defect are associated with symmetrical paralysis of both sides of the body from birth or any early age. These cases of "cerebral diplegia," or Little's disease, are sometimes ascribed erroneously to birth injury. Normally the condition is not progressive. There is a variety of types, and it is certain that some of them are recessive characters. There are also many more conditions inherited in the same way and often associated with severe mental impairment. Among these are some types of cretinism, deafmutism, congenital eye defects and extreme underdevelopment of the brain (microcephaly). Cases of severe mental defect, which are genetic in origin and yet are not due to recessive factors, form another group. Their occurrence is, for the most part, sporadic; that is to say, they occur unexpectedly in normal families. One curious disease, in which the brain is malformed in a manner which, according to the pathologists, resembles potato roots (tuberose sclerosis) and in which tumors develop on the skin and in other parts of the body, often causes severe mental impairment and epilepsy.—L. S. Penrose, in *Sci. Monthly*, April, 1941.

ON THE INHERITANCE OF EAR PIT

(Fistula Auris Congenita) with Special Reference to Twins

E. S. McDonough
Marquette University, Milwaukee, Wisconsin

ONE-EGG TWINS—ONE HAS AN EAR PIT AND ONE DOES NOT

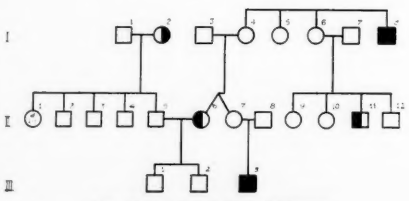
Figure 12

The twin not having the fistula has a son who has one. Her sister who has an ear pit has two children, neither of whom shows the trait. See Figure 13. At the right is an ear of another member of the family (I-8 in Figure 13), showing the location of the pit in this family.

REPORTS by Whitney,[4] Quelprud,[5] and Stiles,[3] have recently emphasized inheritance of ear pit, or *Fistula auris congenita*, as an irregular dominant. Quelprud has reviewed some of the many reports of this condition. The pedigree presented below should throw further light on the production of this interesting trait.

A typical example of the fistula as found in some members of the family whose pedigree is here presented is shown in Figure 12. Individual differences were, however, observed. In the individual represented by Figure 12 (I-8) a pit was found to be located on the superior, posterior portion of the helix of each ear. The fistulae were blind pits a few millimeters in depth similar to the one shown in Figure 12. The other members of the family had pits which were located on the anterior side of the helix. The trait was bilateral (Figure 13; I-8 and III-3) or unilateral (Figure 13; I-2, II-6, and II-11). Figures 12 and 13 (II-6 and 7) represent twin sisters. That these are one-egg twins is indicated by Figure 12 (left). Although these individuals have for some years lived apart their physical characteristics and early history indicate their common origin. Both react positively to P.T.C. taste reaction. They are very similar in eyelashes, eyebrows, lips, shape of ears, hair color, skin color, finger shape and size, and eye color. Although one of the pair has had her teeth removed the best evidence indicates that the teeth were very

similar. They are both right handed. The fistula as found in the one twin is several millimeters deep and discharges a milky substance. At times this region becomes irritated. The other twin has no indication of a pit but her son has fistulae similar in many respects to that of his aunt. His pits are somewhat shallower and discharge less freely.



AN EAR PIT PEDIGREE

Figure 13

The individual represented by I-8 has a fistula located on the superior, posterior portion of the helix of each ear. All others with pits have them at the anterior margin of the helix. I-8 and III-3 have both ears involved. In I-2 only the right ear has a pit. A fistula is located on the left side in II-6 and II-11. II-6 and II-7 are one-egg twins shown in Figure 12 (left).

Discussion

Many published pedigrees have tended to show that *Fistula auris congenita* is inherited as a dominant. The reports of Kindred,[2] Whitney,[4] Quelprud,[5] and Stiles,[3] have indicated that the character may skip a generation. This has led to the assumption that the gene is an irregular dominant. It is perhaps significant that in the instances of one-egg twins reported by Brauns,[1] Quelprud,[5] and the author, only one member of each of the six pairs possessed a pit. In only one instance was there evidence of concordance. One of a pair was reported to have a pit and the other a tumor-like formation which might have been a closed cyst. This, coupled with the frequent occurrence of the unilateral condition in many families would suggest a connection of this trait with the asymmetry pattern. That the twin not showing a fistula has a factor tending to produce the pit seems to be indicated by Figure 13. The presence of the character in the individual represented by III-3 suggests that he has inherited the potentiality for the condition from his mother who did not possess the trait. The necessity of assuming an unstable gene or chromosome irregularity in the families in which a variable effect of the gene is indicated is obviated. That the factor tending to produce the condition is more potent in certain genotypes is of course a possibility. It is also a possibility that there is an effect of pre-natal environment on the time of gene action. Further investigation of the hereditary background should be made.

Summary

A pedigree of a family in which *Fistula auris congenita* occurs is presented involving a set of one-egg twins. The mode of transmission was considered to support the idea that the trait was produced under the influence of an irregular dominant. The asymmetry pattern and pre-natal environment were thought to affect the nature and presence or absence of the pits.

Literature Cited

1. Brauns, L. Studien an Zwillingen. Säuglings-und Kleinkindesalter. *Zeitschr. f. Kinderforschung* 43:86-129. 1934.

2. Kindred, J. E. Inheritance of a Pit in Skin of the Left Ear. *Jour. Hered.* 12:366-367. 1921.

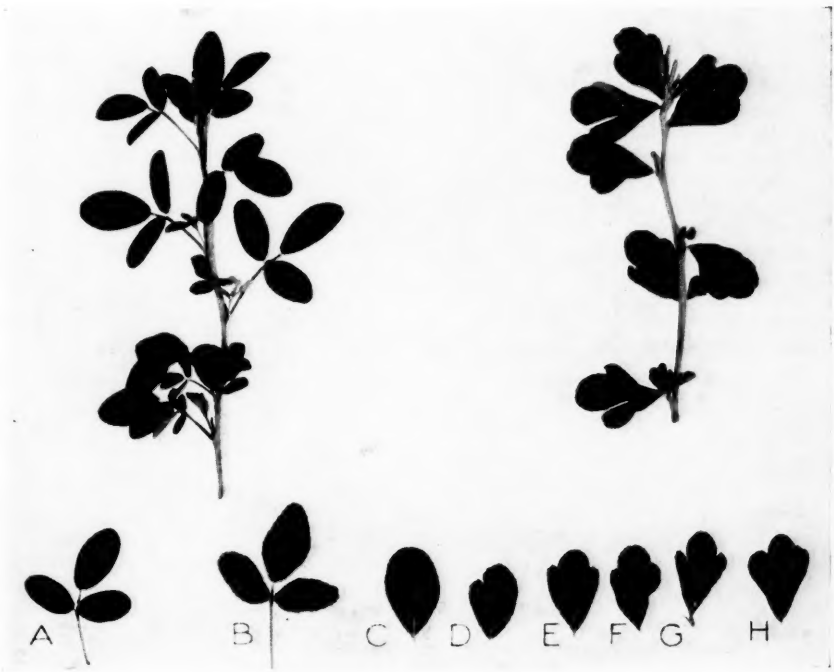
3. Stiles, K. A. The Inheritance of Pitted Ear. *Genetics* 26:171. 1941. (Abstract.)

4. Whitney, D. D. Three Generations of Ear Pits. *Jour. Hered.* 30:322-324. 1939.

5. Quelprud, T. Ear Pit and Its Inheritance. *Jour. Hered.* 31:379-384. 1940.

A NEW MUTANT LEAF CHARACTER IN SWEET CLOVER*

E. E. HARTWIG†



NORMAL AND "ONE-LEAVED" CLOVER

Figure 14

At the left a branch and leaves (*A-B*) of a normal sweet clove plant are shown. At the right is a branch of the unifoliolate mutation, and below it a series of leaves (*C-H*) show the range of variation of the character.

A DEVIATION from the normal trifoliolate leaf of sweet clover has appeared in the first selfed generation of a selection of *Melilotus officinalis*. The mother plant was selected from an introduction of the U. S. Department of Agriculture, P. I. 31647, from India, all plants of which had normal leaves. The plant which gave rise to the mutant type was selected and bagged to obtain selfed seeds in the summer of 1937 as a part of the sweet clover improvement program.

The first generation selfed seeds were planted in flats in the greenhouse in the spring of 1938 and the seedlings were later transplanted to the field nursery. Soon after the seedlings developed leaves, a portion of the plants was observed to have an abnormal unifoliolate

*Contribution from the Division of Forage Crops and Diseases, Bureau of Plant Industry, U. S. Department of Agriculture, and the Department of Agronomy, Illinois Agricultural Experiment Station.

†Agent, Division of Forage Crops and Diseases, Bureau of Plant Industry, U. S. Department of Agriculture.

leaf type. Of a population of 49 plants, 38 had the normal trifoliolate leaf and 11 had the new unifoliolate leaf, a very close approximation to a 3:1 ratio.

The leaves of the unifoliolate plants are nearly sessile and vary in shape from a single leaf resembling an enlarged central leaflet of a trifoliolate leaf to various degrees of lobing. Figure 14 illustrates the normal leaf pattern and the variations in leaf shape on the mutant type. Atwood[1] has described a somewhat similar character on a single plant of white clover (*Trifolium repens*). Lamprecht[2] found that a single leaf character in *Pisum* was inherited as a simple recessive and was closely linked with an abnormal flower condition. The sweet clover plants with unifoliolate leaves have all had normal floral development.

The unifoliolate type has been reproduced through three generations by selfing and sibbing. Reciprocal crosses with the normal leaf type have given F_1 plants with normal leaves. F_2 populations have been grown from two crosses in which the unifoliolate type was used as the female parent. Table I gives the results of these crosses.

The results obtained from growing the original segregating population and the two F_2 progenies indicate that the mutant leaf type differs from the normal by a single recessive gene.

Literature Cited

1. Atwood, S. S. A "one-leaved" white clover. *Jour. Hered.* 29:239-240. 1938.

2. Lamprecht, H. Ein unifoliata-typus von Pisum mit gleichzeitiger Pistilloidie. *Hereditas* 18:56-64. 1934.

TABLE I. Numbers of trifoliolate and unifoliolate plants in two segregating F_2 progenies, with numbers expected on the basis of a 3:1 ratio and values of Chi-square.

Number of plants	Observed		Expected		
	Trifoliolate	Unifoliolate	Trifoliolate	Unifoliolate	X^2
23	18	5	17.25	5.75	0.130
38	34	4	28.50	9.50	4.246
			Degrees of freedom — 2 P between .10 and .20		4.376

◆

The "Dresden," a Psycho-Lethal

ALL of which brings up another point about these eastern apartment-house women who suddenly find themselves in Reno. They are not quite ready for the big jump into desert loneliness. They want their town and their wilderness, too. So accustomed all their lives to city crowds, these women must taper off gradually if they taper off at all. They must know that the heart of the town is right there, within ten or fifteen minutes, if they need it of an evening. And if they are to have dates.

For few of these women who come to Reno have their own cars. They can rent cars, of course, in Reno. But this runs into money, it over-steps their divorce budget. It is better to make a date with somebody who has a car. But no man who owns a car is especially anxious to drive twenty or more miles one way to get the date, twenty or more miles to get her back to town again for the spots, then twenty miles to deliver her to the ranch in the early morning, then twenty miles back to town again —just in time for him to go to work. He may be willing enough, naturally, if there is something it in for him. But how is he to know ahead of time?

She may have all the looks, she may have all the come-on in her voice, she may have all the acted-out warmth. But underneath it all she may be what the men of Reno call a "Dresden," a derivation of "Dresden Doll" — meaning that her looks are her whole life, her profession, and that in no way does she want to endanger that china body of hers either by masculine handling or by cacti or by pregnancy.

And this latter danger, so the Reno lawyers tell us, may be exactly the reason her husband has shipped her off for the six weeks. Next time, for the sake of his future lineage, he will choose a peasant. Or maybe he already has done so.

Yes, the males of Reno, especially the lawyers, have had a good many years of practice now in grading the women of this great nation of ours. Endlessly, ever since the turn of the century, they have been passing through Reno as on a conveyor-belt. The Dresden-faced beauties who annually are sent to Reno may be supreme in novels, but in bed they still remain the handle-me-with-care-use-no-hooks dolls they are. And the husbands have had enough of them.

Even in conversation she usually has nothing. Her whole religion is her looks, nothing else, the turn of her eyelashes, her fingernails, the lobe of her ear, and that valued though useless waistline. — Max Miller, in *Reno's Dude Ranches*, Dod-Meade & Co., 1941.

THE INHERITANCE OF THREE COAT COLOR MUTATIONS IN RANCH-RAISED MINKS

SEDGWICK E. SMITH,* C. H. WHITAKER,† LEO F. DAVIDS‡ AND PERCY V. NOBLE§

COAT color mutations in fur animals, in addition to having genetic interest, may have great economic value such as the case of the Norwegian platinum fox described in 1939 by Mohr and Tuff.[2] Fur animal breeders are usually aware of this and when a color mutation appears it is carefully kept and reproduced. Such animals are usually priced beyond the budget of most geneticists and therefore it is necessary to secure the cooperation of the breeders for genetic analyses. This has been the case in this study where careful breeding records have permitted an analysis of the inheritance of three mutations which are called platinum, blond, and silver.

The wild mink (*Mustela vison*), which is the progenitor of the ranch-raised mink, possesses dark brown guard hairs of various shades and a lighter brown underfur. Ranch-raised minks have been selectively bred to meet the demands of the consumer for a dark chocolate-brown guard hair and a blue-gray underfur. The dark guard hairs and the blue-gray underfur give the pelt a desirable contrast of colors. The wild type mink also possesses various but small amounts of white spotting on the abdomen and chin, and its distribution is often made the basis of identification of individual minks. Figure 15*A* is a picture of a show champion, wild type mink.

The Platinum Mink

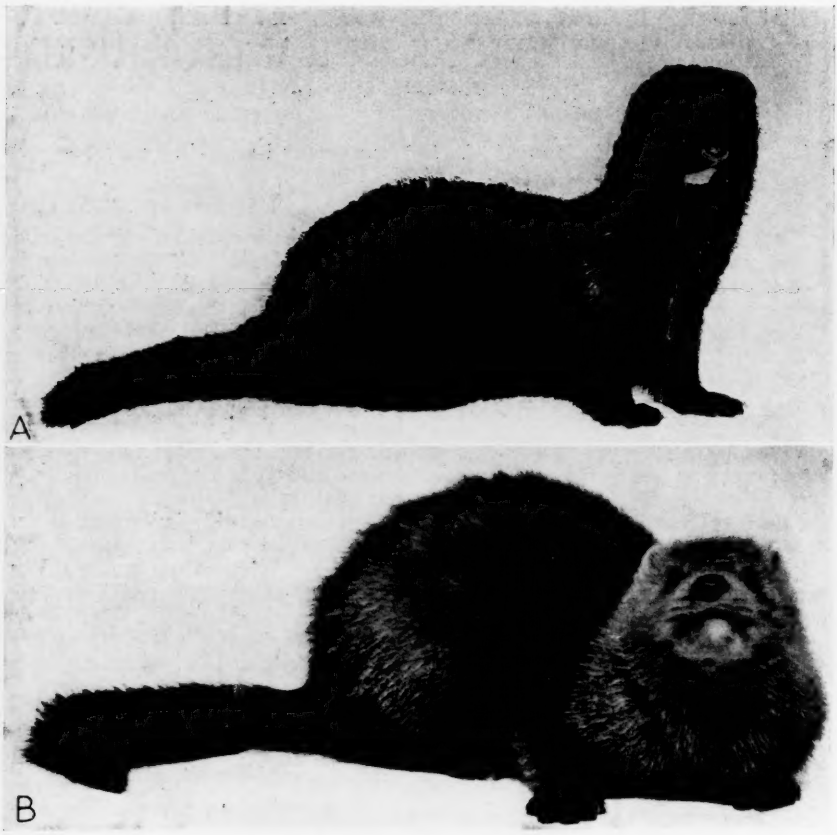
In the spring of 1938 two very pale female mink kits appeared in a litter of seven, born from a mating of wild type minks in the herd of one of the junior authors (C. H. W.). These two kits developed into the so-called platinum minks (Whitaker strain). A typical platinum mink possesses light pearl-gray guard hairs which are somewhat lighter on the abdomen and darker at the extremities—tail and feet (Figure 15*B*). The nose is lighter than the liver-colored wild type, and the eves are lighter than the dark brown wild type. The underfur is a pale blue-gray color.

A phenotypically similar and genetically identical mutation appeared in the herd of Mr. Whittingham of Arpin, Wisconsin, "about 10 years ago." This strain is being developed by Mr. Whittingham and Mr. Ingham to whom the authors are indebted for some additional information.

The Blond Mink

Two litters of three and of two kits were born of wild type minks in the herd of one of us (L. F. D.) in the spring of 1937. In the former litter were two female kits and in the second one female kit of a color lighter than normal. These three kits were the first blond minks. The prime fur of a blond mink is made up of light brown guard hairs and a pale blue-gray underfur. The sun striking the prime pelt gives it an attractive metallic sheen. The noses of blond minks vary from pink to a light liver-color in contrast to the much darker wild type. Likewise, the wild type eye is diluted to a lighter shade. There are light and dark phases of the blond mink but in no case has the color overlapped that of the wild

*Agent, U. S. Bureau of Animal Industry and research instructor in the Laboratory of Animal Nutrition, Cornell University. Formerly cooperating with the Fish and Wildlife Service, U. S. Department of the Interior.
†Union Grove, Wisconsin.
‡Geneva, New York.
§Shallow Lake, Ontario, Canada.



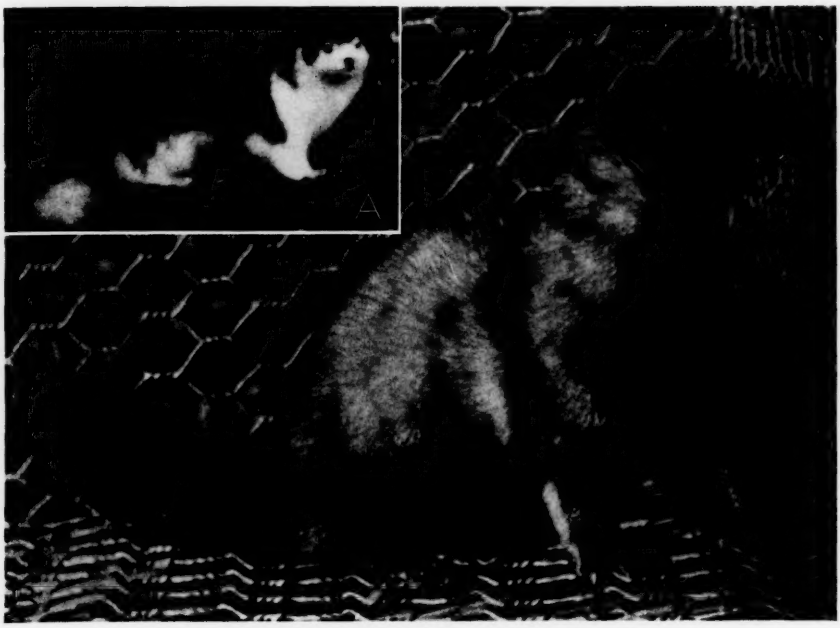
NORMAL AND PLATINUM MINKS
Figure 15

A—A prize-winner in the mink prize ring. The guard hairs of the most desirable type of ranch-raised mink are dark chocolate brown, with blue-grey underfur. *B*—A platinum mink. In 1938 two kits in a litter of seven had fur of this color, and a genetically identical mutation has since been found which occurred in another herd about ten years ago. The platinum mutation appears to have definite economic importance, as a coat made of 39 platinum pelts has recently been offered for sale for $39,000. Photographs courtesy of the *American Fur Breeder*.

type from which it is easily distinguished, as the senior author has personally observed. One of the light blond minks is shown in Figure 16*B*.

The Silver Mink

A single silver and white spotted male kit was born of wild type minks in the herd of the third junior author (P.V.N) in the spring of 1937. This kit matured as pictured in Figure 16*A*. The white spotting was much more extensive than ever found in the wild type. The pigmented hairs were intermixed with the white ones giving a silver effect. The white spotted and silver offspring which resulted from the mating of this original male with wild type females have varied much in the extent of white spotting and in silvering. In some cases the silver offspring resemble the platinum fox.



BLOND AND SILVER MINKS

Figure 16

A—The original male silver mink discussed in this article. Offspring of this male have been variously spotted, some of them resembling platinum foxes. *B*—A light phase blonde mink. The guard hairs are light brown and the underfur is pale gray. A darker phase can always be distinguished from the normal coat color.

Breeding Results

A summary of the various matings involving platinum, blond, silver, and normal colored minks is given in Table I. It is evident that platinum and blond are each inherited as simple recessive characters, and that silver is a simple dominant character. The observed ratio of 60 normals : 46 platinums which resulted from the mating of platinum × heterozygotes is a good fit for a 1 : 1 ratio (D/P.E. 2.01) as well as the observed ratio of 14 normals : 6 blonds which resulted from blond × heterozygotes (D/P.E. 2.64).

The observed ratio of 32 silvers : 16 normals which resulted from the mating of silver × silver is obviously a good fit for a 3 : 1 ratio.

That platinum is autosomal and not sex-linked was shown by the matings of a normal colored male with two platinum females. The offspring consisted of four platinum females and three normal colored males. Had the character been sex-linked all male offspring would have been platinum colored.

The evidence that blond is autosomal was derived from two different matings of normal colored males × normal colored females. The offspring were made up of three blond females and two normal colored males which showed that both parents carried the blond factor. Had the character been sex-linked the male parents would have been blond instead of normal color.

That the silver factor is not sex-linked was shown in eighteen different matings of silver males × normal colored fe-

males. The distribution of coat color according to the sex of the progeny was 22 normal colored males : 19 silver males : 16 normal colored females : 17 silver females. Had the factor been sex-linked all male offspring would have been normal colored and all female offspring silver.

That the platinum mutation of the Whitaker strain is genetically identical to the platinum mutation of the Whittingham-Ingham strain was shown by a cross of a female platinum of the former and a platinum male of the latter in which two platinum kits were produced.

The matings involving the silver factor have not been extensive enough yet to test the offspring of silver × silver to find the homozygous dominant individuals if they exist. Questions relative to the viability and appearance of such animals must await more extensive breeding results.

The mutants as far as observed are as vigorous and healthy as normal ranch-raised minks. Eight litters derived from platinum × platinum averaged 3.5 kits per litter, 7 litters from blond × blond averaged 4.1 kits, and 14 litters from silver × silver averaged 3.7 kits. These numbers are small but they suggest that fertility is comparable to that of normal minks where the average number of kits per litter is about four.

Discussion

The factors bringing about the development of the platinum and the blond colors may be classed as dilution factors, the platinum being more extreme than the blond. A knowledge of the relationship of these two factors would be interesting. The fact that the extremities of platinum minks are darker than the remaining parts of the body suggests a similarity to the Himalayan (c^H) factor in rabbits and and the Siamese factor in cats.

The silver factor is somewhat like the silver factor in mice[1] in that there is an intermixture of pigmented and non-pigmented hairs. However, in the silver mouse there is no associated white spotting as there is in the silver mink.

That at least one of these mutants is destined for a profitable future is indicated by the recent completion of a box coat by a New York furrier. The coat was made of 39 platinum mink pelts and is being offered for $39,000.

Table I. Results of matings involving platinum, blond, silver, and normal (brown) minks.

Type of cross	Observed ratio		Expected ratio		Number of matings
	Normal	Platinum	Normal	Platinum	
Platinum X normal (pp X PP)	19	0	19	0	5
Platinum X platinum (pp X pp)	0	28	0	28	8
Platinum X heterozygote* (pp X Pp)	60	46	53	52	23
Heterozygote X heterozygote* (Pp X Pp)	29	7	27	9	5
	Normal	Blond	Normal	Blond	
Blond X normal (bb X BB)	19	0	19	0	4
Blond X blond (bb X bb)	0	29	0	29	7
Blond X heterozygote* (bb X Bb)	14	6	10	10	5
	Silver	Normal	Silver	Normal	
Silver X normal (Ss X ss)	36	38	37	37	17
Silver X silver** (Ss X Ss)	35	16	38.2	12.8	14

* The above heterozygotes were normal (brown) minks one of whose parents was a mutant.
** One of the parents of each silver mink was a normal colored mink.

Summary

Three coat color mutations from wild type minks have been described. The pearl-gray mutation, called platinum (p), is inherited as a simple, autosomal recessive character. The dilute brown mutation (b), called blond, is also inherited as a simple, autosomal recessive character. The third character, silver (S), is inherited as a simple, autosomal dominant. Judgment as to the nature of the homozygous dominant silver mink must be withheld until a greater number of matings are made.

The mutant minks are vigorous, healthy, and fertile.

The writers are grateful to Dr. F. B. Hutt, Cornell University, for reading the manuscript.

Literature Cited

1. Dunn, L. C. and Thigpen, L. W. The silver mouse. A recessive color variation. *Jour. Heredity* 21:495-498. 1930.

2. Mohr, Otto L., and Tuff, Per. The Norwegian platinum fox. A coat color mutation having great economic value. *Jour. Heredity* 30:227-234. 1939.

Revised — Enlarged

Genetics and the Origin of Species

By Theodosius Dobzhansky

In its original form this work has been widely circulated and acclaimed. This new, up-to-date edition takes into consideration all progress made and new developments that have taken place since the completion of the manuscript of the first edition. No student of genetics can afford to ignore it.

465 pages, illustrated
$4.25

Columbia University Press
Morningside Heights, N. Y.

SALIVARY GLAND CHROMOSOMES

A Symposium Reprint

Comprising articles on Salivary Gland Chromosomes in the JOURNAL OF HEREDITY, 1934-1940, by Painter, Bridges, Mackensen, Metz, Hughes. Four Salivary Gland Chromosome maps, 15 illus., 54 pages. $3.

American Genetic Association
Victor Building,
Washington, D. C.

INBRED MICE

FOR RESEARCH IN EXPERIMENTAL BIOLOGY AND MEDICINE

DURING the past few years this Laboratory has completed the establishment of a number of inbred and hence genetically homogeneous strains of mice which have proven extremely valuable for research in experimental medicine and biology. The inbreeding is carried on through repeated brother times sister matings. The essential consequence of the inbreeding is that the individuals within a strain become increasingly uniform in their heredity and after twenty generations may be regarded for all practical purposes as genetically identical. The use of such material largely eliminates a common source of difficulty in biological investigations, namely, the inherent variability of the organisms ordinarily available. The uniformity of inbred strains is illustrated by the fact that tumors originating in such a strain, when transplanted. will grow in 100% of the individuals of that strain, but will grow irregularly if at all in other strains.

Careful checks are maintained on the homogeneity of the strains carried by this Laboratory. Sublines tend to develop within an inbred strain due to the accumulation of minor mutations. These are detected and where advisable eliminated through the use of appropriate tests and breeding methods.

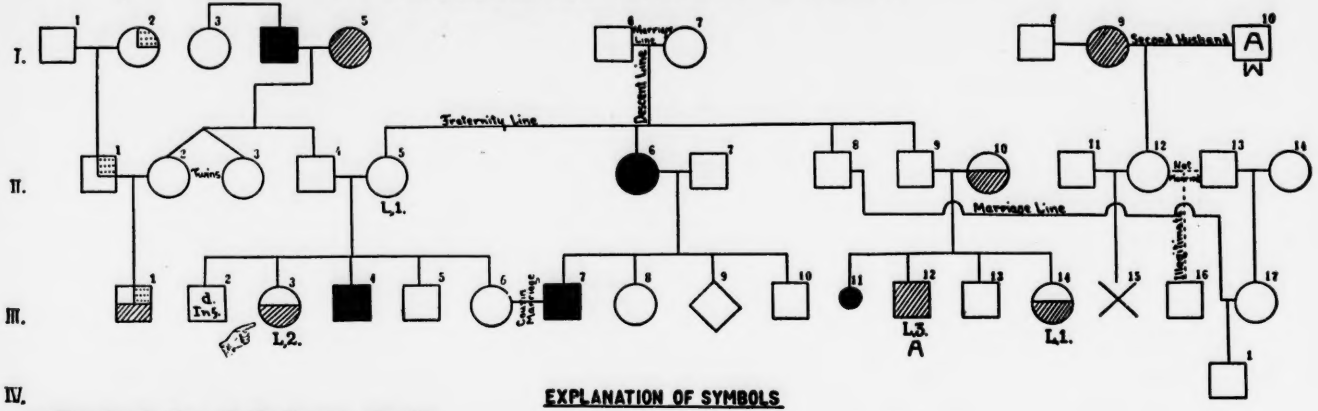
The Laboratory will welcome inquiries from scientific laboratories and hospitals concerning the inbred stock available or with regard to the possibility of providing stocks and types suitable for special experimental problems.

Supply Department

ROSCOE B. JACKSON MEMORIAL LABORATORY

Bar Harbor **Maine**

SAMPLE PEDIGREE CHART SHOWING THE MANNER OF CONSTRUCTION, AND THE USE OF STANDARD AND SPECIAL SYMBOLS.



EXPLANATION OF SYMBOLS

(a). Standard for all Pedigree Charts:

□=Male; ○=Female; ◇=Sex unknown; ●=Still-birth or Miscarriage; X=Children — number and sex unknown; ○○=Twins; d. Inf.=Died in Infancy; ☞ Points to the Propositus or central figure in the pedigree.

□—○=Parents.

□○□○○=Children.

Roman figures to the left indicate generations; Arabic figures locate individuals, (thus III. 7 is the young man in the third generation who married his cousin).

The following letters, placed in or around the individual's pedigree symbol, are standard for certain traits: A, alcoholic; B, blind; D, deaf; E, epileptic; F, feebleminded; I, insane; M, migraineous; N, normal in reference to traits under consideration; Ne, neurotic; P, paralytic; Sx, sexually immoral; S, syphilitic; T, tuberculous; W, wanderer.

(b). Especially devised or selected for this particular pedigree-chart:

■ ● = Successful leaders in politics. ◱ ◔ = Extra thumb on right hand. L.1.=Highly successful author. L.3.=Little or no ability in literary efforts. ▨ ◍ = Superior in vocal music. ▨ ◍ = Medium attainment in vocal music.

To fit the particular family and traits (whether physical, mental or temperamental; good or bad) under consideration, invent special symbols, or select special letters (in addition to those standardized under (a) above) to be placed within or near the particular individual's pedigree symbol, to indicate particular traits and their degree of development.

Prepared by the International Commission of Eugenics. See *Journal of Heredity* for August 1926. Additional copies may be obtained from A.G.A.